Table 12: **gp160**

	MAb ID	HXB2 Location	Author's Location	Sequence	Neutral-izing	Immunogen	Species (Isotype)
262	M85	gp160(30–51)	gp120(30–51 LAI)	ATEKLWVTVYYGVPVWKEAT-TT	no	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: C1 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Ditzel (1997), Wyatt (1997)]

- M85: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)]
- M85: C1 domain – mutation 40 Y/D impairs binding – the relative affinity for denatured/native gp120 is < .01, suggesting conformational component [Moore (1994c)]
- M85: Binding inhibited by MAb 4D4#85, enhanced by conformationally sensitive anti-V3 MAb 5G11, and some anti-18 MAbs [Moore & Sodroski(1996)]
- M85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)]

263	7E2/4	gp160(31–50)	gp120(31–50 LAI)	TEKLWVTVYYGVPVWKEATT		Vaccine	murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Ab type: C1 **Donor:** S. Ranjbar, NIBSC, UK
References: [Moore (1994c)]

- 7E2/4: C1 domain – the relative affinity for denatured/native gp120 is .07, suggesting conformational component [Moore (1994c)]
- 7E2/4: UK Medical Research Council AIDS reagent: ARP3050

264	4D4#85	gp160(41–50)	gp120(LAI)	GVPVWKEATT		Vaccine	murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **Donor:** S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Wyatt (1997), Binley (1998)]

- 4D4#85: C1 domain – the relative affinity, denatured/native gp120 is 0.1 – mutation 45 W/S impairs binding [Moore (1994c)]
- 4D4#85: Inhibits binding of C1 MAb M85, C1-C5 discontinuous epitope MAbs 181 and 212A, and CD4 binding induced MAbs 48d and 17b [Moore & Sodroski(1996)]
- 4D4#85: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding – does not bind to HXBc2 gp120 if the 19 C-term amino acids, in conjunction with C1 positions 31–50, are deleted [Wyatt (1997)]
- 4D4#85: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

265	M92	gp160(41–50)	gp120(31–50 LAI)	GVPVWKEATT	no	Vaccine	rat(IgG1)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: C1 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]

- M92: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]
- M92: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

266	M86	gp160(42–61)	gp120(42–61 LAI)	VPVWKEATTTLFCASDAKAY	no	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: C1 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c)]

- M86: Immunoblot and RIP reactive for strains IIIB, 451, MN, RF, and RUTZ – binds deglycosylated gp120 [di Marzo Veronese (1992)]
- M86: C1 domain – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

267	polyclonal	gp160(51–70)	Env(42–61 LAI)	LFCASDAKAYDTEVHNVWAT	no	Vaccine	murine( )

Vaccine: *Vector/type:* vaccinia *HIV component:* Env

Ab type: C1 **References:** [Collado (2000)]

- Vaccinia p14 can elicit NAbs and p39 tends to be immunodominant, so these two proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCASDAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)]

268	133/237	gp160(61–70)	gp120(51–70 LAI)	YDTEVHNVWA	L	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **References:** [Niedrig (1992b), Moore (1994c), Moore (1994d)]

- 133/237: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)]
- 133/237: The relative affinity, denatured/native gp120 is 1.4 – mutation of position 69 W/L impairs binding [Moore (1994c)]

269	133/290	gp160(61–70)	gp120(61–70 LAI)	YDTEVHNVWA	L	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **References:** [Niedrig (1992b), Thali (1993), Moore (1994c), Moore (1994d), Wyatt (1995), Binley (1997a), Wyatt (1997), Binley (1998)]

- 133/290: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)]
- 133/290: The relative affinity for denatured/native gp120 is 2.2 – mutation in position 69 W/L impairs binding [Moore (1994c)]

- 133/290: Used for antigen capture assay, either to bind gp120 to the ELISA plate, or to quantify bound gp120 [Wyatt (1995)]
- 133/290: Reciprocal binding inhibition with the antibody 522–149, that binds to a discontinuous epitope – binding is enhanced by some C5 and C1 binding site antibodies [Moore & Sodroski(1996)]
- 133/290: Binds efficiently to sgp120 but not soluble gp120+gp41, suggesting its gp120 epitope is blocked by gp41 binding [Wyatt (1997)]
- 133/290: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

---

270 133/11 gp160(64–78) gp120(64–78) EVHNVWATHACVPTD L Vaccine murine(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **References:** [Niedrig (1992b)]

- 133/11: Region of overlap for reactive peptides is WATHA – weak neutralization of lab strains [Niedrig (1992b)]

---

271 D/3G5 gp160(73–82) gp120(73–82 LAI) ACVPTDPNPQ no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/3G5: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

272 D/6A11 gp160(73–82) gp120(73–82 LAI) ACVPTDPNPQ no Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/6A11: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

273 D/5E12 gp160(73–92) gp120(73–92 LAI) ACVPTDPNPQEVVLVNVTEN no Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/5E12: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

274 L5.1 gp160(79–93) gp120(89–103 IIIB) PNPQEVVLVNVTENF Vaccine murine(IgG)

Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* gp160

Ab type: C1 **References:** [Akerblom (1990)]

---

275 4A7C6 gp160(81–90) gp120(81–90 LAI) PQEVVLVNVT Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Ab type: C1 **Donor:** R. Tedder
References: [Thiriart (1989), Thali (1993), Moore & Ho(1993), Moore (1994c), Moore (1994d), Moore & Sodroski(1996)]

- 4A7C6: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 4A7C6: The relative affinity for denatured/native gp120 is 7.9 – mutation 88 N/P impairs binding [Moore (1994c)]
- 4A7C6: C1 region epitope (88 N/P substitutions abrogates binding), but substitutions 380 G/F and 420 I/R also impaired binding [Moore (1994d)]
- 4A7C6: Reciprocal binding inhibition with the antibody 133/192 – enhanced by anti-C5 antibodies, and C1 antibody 135/9[Moore & Sodroski(1996)]
- 4A7C6: UK Medical Research Council AIDS reagent: ARP 360

---

276 1D10 gp160(81–100) gp120(81–100 LAI) PQEVVLVNVTENFDMWKNDM L Vaccine rat( )

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **References:** [Dowbenko (1988), Berman (1991), Nakamura (1992), Moore (1994c)]

- 1D10: Cross-blocks 5B3 in IIIB-rsgp160 ELISA – type specific in rgp120 ELISA binding [Nakamura (1992)]
- 1D10: The relative affinity for denatured/native gp120 is 13 – mutation 88 N/P impairs binding [Moore (1994c)]

---

277 B242 gp160(83–92) gp120(83–92 LAI) EVVLVNVTEN no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* NL43 *HIV component:* gp160

Ab type: C1 **References:** [Bristow (1994)]

- B242: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)]

---

278 133/192 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM L Vaccine murine(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **Donor:** Matthias Niedrig
References: [Niedrig (1992b), Moore (1993b), Moore (1994c), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Binley (1998)]

- 133/192: Epitope seems complex, binds multiple peptides – weak neutralization of lab strain [Niedrig (1992b)]
- 133/192: The relative affinity for denatured/native gp120 is 1.8 [Moore (1994c)]
- 133/192: C1 region – substitutions 76P/Y, 113 D/A or R, 117 K/W, 420 I/R, 427 W/S impair binding, other substitutions enhanced binding [Moore (1994d)]
- 133/192: Reciprocal binding inhibition with the antibody 4A7C6 – enhanced by some anti-C5 and-C1 antibodies [Moore & Sodroski(1996)]
- 133/192: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]

- 133/192: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

---

279 489.1(961) gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **Donor:** C. Bruck, SKB, Belgium
References: [Moore (1994c)]

- 489.1(961): C1 region – The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]
- 489.1(961): NIH AIDS Research and Reference Reagent Program: 961

---

280 5B3 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM no Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp160

Ab type: C1 **References:** [Berman (1991), Nakamura (1992), Beretta & Dalgleish(1994), Moore (1994c)]

- 5B3: Blocks gp120 -CD4 binding [Berman (1991)]
- 5B3: Cross-blocks 1D10 in competitive IIIB-rsgp160 ELISA – no neutralization – blocks IIIB-gp120 sCD4 binding – localized binding to residues 72–106 [Nakamura (1992)]
- 5B3: The relative affinity of denatured/native gp120 is 8.3 [Moore (1994c)]

---

281 B10 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994), Moore (1994c)]

- B10: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- B10: The relative affinity for denatured/native gp120 is 0.4 [Moore (1994c)]
- B10: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)

---

282 B2 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Thali (1993), Abacioglu (1994), Moore (1994c), Moore (1994d), Binley (1997a)]

- B2: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- B2: The relative affinity for denatured/native gp120 is 1.4 [Moore (1994c)]
- B2: There is FNM/FDM polymorphism in LAI-based peptides, and N is essential (J. P. Moore, per. comm.)

---

283 C6 (Ch6) gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Pincus & McClure(1993), Abacioglu (1994), Moore (1994c), Pincus (1996)]

- C6: C1 region – epitope boundaries mapped by peptide scanning, FNMW core [Abacioglu (1994)]
- C6: The relative affinity for denatured/native gp120 is 0.9 [Moore (1994c)]

- C6: There is FNM/FDM polymorphism in LAI-based peptides – N is essential (J. P. Moore, per. comm.)
- C6: Called Ch6 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]
- C6: NIH AIDS Research and Reference Reagent Program: 810

---

284 MF49.1 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF49.1: The relative affinity of denatured/native gp120 is 3.8 [Moore (1994c)]

---

285 T1.1 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG)

Vaccine: *Vector/type:* vaccinia *HIV component:* gp160

Ab type: C1 **References:** [Akerblom (1990), Broliden (1990), Moore (1994c)]

- T1.1: Also reacted in solid phase with gp120(234–248) NGTGPCTNVSTQCT [Akerblom (1990)]
- T1.1: No ADCC activity – reactive peptide: NVTENFNMWKNDMVEQ, IIIB [Broliden (1990)]
- T1.1: C1 region – the relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

---

286 T7.1 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)]

- T7.1: The relative affinity of denatured/native gp120 is 4.0 [Moore (1994c)]

---

287 T9 gp160(91–100) gp120(91–100 LAI) ENFDMWKNDM Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **Donor:** Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d), Binley (1997a)]

- T9: The relative affinity of denatured/native gp120 is 7.9 [Moore (1994c)]
- T9: C1 region – 45 W/S, 88 N/P, 256 S/Y, 262 N/T, 475 M/S, 485 1.83, and 491 I/F enhanced binding, no substitution tested significantly inhibited [Moore (1994d)]

---

288 GV4D3 gp160(92–100) gp120(92–100 IIIB) NFNMWKNDM Vaccine murine( )

Vaccine: *Vector/type:* protein-Ab complex *HIV component:* gp120 complexed with MAb M77

Ab type: C1 **References:** [Denisova (1996)]

- GV4D3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV4H4 and GV5F9 are homologous to GV4D3 and were generated in the same experiment [Denisova (1996)]

---

289 B27 gp160(93–96) gp120(94–97 BH10) FNMW no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* NL43 *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994), Bristow (1994)]

- B27: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]
- B27: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)]

---

290 B9 gp160(93–96) gp120(93–96 LAI) FNMW Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994)]

- B9: Binds C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

291 B35 gp160(93–98) gp120(94–99 BH10) FNMWKN Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994)]

- B35: C1 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

292 D/4B5 gp160(93–101) gp120(93–101 LAI) FNMWKNDMV no Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/4B5: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

293 D/5A11 gp160(93–101) gp120(93–101 LAI) FNMWKNDMV no Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/5A11: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

294 D/6B2 gp160(93–101) gp120(93–101 LAI) FNMWKNDMV no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: C1 **References:** [Bristow (1994)]

- D/6B2: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

295 B18 gp160(101–110) gp120(101–110 LAI) VEQMHEDIIS Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994), Moore (1994c)]

- B18: C1 region – epitope boundaries mapped by peptide scanning, HEDII core [Abacioglu (1994)]
- B18: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

---

296 B20 gp160(101–110) gp120(101–110 LAI) VEQMHEDIIS Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **References:** [Abacioglu (1994), Moore (1994c)]

- B20: C1 region – epitope boundaries mapped by peptide scanning – HEDII core [Abacioglu (1994)]
- B20: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]

---

297 MF39.1 (39.1) gp160(101–110) gp120(101–110 LAI) VEQMHEDIIS Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Cook (1994), Moore (1994c)]

- MF39.1: Called 39.1, and is probably the same as MF39.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]
- MF39.1: The relative affinity of denatured/native gp120 is 30 [Moore (1994c)]

---

298 187.2.1 (187.1) gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Ab type: C1 **Donor:** Claudine Bruck and Clothilde Thiriart
References: [Thiriart (1989), Moore & Ho(1993), Cook (1994), Moore (1994c), Moore (1994d)]

- 187.2.1: Called 187.1, and is probably the same as 187.2.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 187.2.1: Called 187.1, and is probably the same as 187.2.1 – MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]
- 187.2.1: The relative affinity for denatured/native gp120 is 7 – mutations 113 D/A (not D/R) and 117 K/W impair binding [Moore (1994c)]
- 187.2.1: UK Medical Research Council AIDS reagent: ARP332

---

299 37.1.1 (37.1) gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Ab type: C1 **Donor:** Claudine Bruck
References: [Thiriart (1989), Moore & Ho(1993), Moore (1994c)]

- 37.1.1: Called 37.1 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]

- 37.1.1: The relative affinity for denatured/native gp120 is 8.6 – mutations 113 D/R (not D/A) and 117 K/W impair binding [Moore (1994c)]
- 37.1.1: UK Medical Research Council AIDS reagent: ARP327

---

300 6D8 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine rat( )

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **References:** [Dowbenko (1988), Nakamura (1992), Moore (1994c)]

- 6D8: Highly cross-reactive with multiple stains by rgp120 ELISA [Nakamura (1992)]
- 6D8: The relative affinity for denatured/native gp120 is 15 – mutations 113 D/R and 113 D/A impair binding [Moore (1994c)]

---

301 M96 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV no Vaccine rat(IgG2a)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: C1 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]

- M96: Immunoblot reactive for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]
- M96: C1 region – the relative affinity for denatured/native gp120 is 6 [Moore (1994c)]

---

302 MF119.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF119.1: The relative affinity for denatured/native gp120 is 30 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore (1994c)]

---

303 MF4.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF4.1: The relative affinity for denatured/native gp120 is 8 [Moore (1994c)]

---

304 MF53.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF53.1: The relative affinity for denatured/native gp120 is 10 [Moore (1994c)]

---

305 MF58.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

---

306 MF77.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF77.1: The relative affinity for denatured/native gp120 is 11 [Moore (1994c)]

---

307 T2.1 gp160(101–120) gp120(101–120 LAI) VEQMHEDIISLWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **Donor:** Lennart Akerblom, Britta Wahren and Jorma Hinkula
References: [Akerblom (1990), Bolmstedt (1990), Moore (1994c), Moore (1994d)]

- T2.1: The relative affinity for denatured/native gp120 is .27 – mutations 113 D/R, 106 E/A, and 117 D/A impair binding [Moore (1994c)]

---

308 11/65 (11/65a/5h) gp160(dis 102–121) gp120(dis 311–321 HXB10) EQMHEDIISLWDQSLKPCVK Vaccine rat(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: C1 **References:** [McKeating (1992a), McKeating (1993b), Peet (1998)]

- 11/65: Binds only soluble gp120, not virion bound – used to quantify gp120 shedding – (numbering is incorrect in original?) [McKeating (1992a)]
- 11/65: Called 11/65a/5h – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/65 was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/65: UK Medical Research Council AIDS reagent: ARP3076

---

309 W1 gp160(102–121) gp120(102–121 LAI) EQMHEDIISLWDQSLKPCVK Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **Donor:** D. Weiner, U. Penn.
References: [Moore (1994c)]

- W1: The relative affinity for denatured/native gp120 is 6 – mutations 113 D/A, 113 D/R, and 117 K/W impair binding [Moore (1994c)]

---

310 T11 gp160(102–125) gp120(102–125) EQMHEDIISLWDQSLKPCVKL-TPL Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *HIV component:* oligomeric gp140

Ab type: C1 **Donor:** R. Doms, Univ. of Pennsylvania
References: [Earl (1994), Jagodzinski (1996)]

- T11: Generated during a study of the influence of the oligomeric structure of Env in determining the repertoire of the Ab response – an oligomer with no gp120/gp41 cleavage site was used as the immunogen [Earl (1994)]

- T11: The sulfated polysaccharide, curdlan sulfate (CRDS), binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent T11 inhibition by CRDS [Jagodzinski (1996)]

---

311 GV1A8 gp160(105–113) gp120(105–113 IIIB) HEDIISLWD Vaccine murine( )

Vaccine: *Vector/type:* protein-Ab complex *HIV component:* gp120 complexed with MAb M77

Ab type: C1 **References:** [Denisova (1996)]

- GV1A8: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MABs to linear epitopes – MAbs GV7A4 and GV5H5 are homologous to GV1A8 and were generated in the same experiment [Denisova (1996)]

---

312 11 gp160(111–120) gp120(101–120 LAI) LWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- 11: The relative affinity for denatured/native gp120 is 7.8 – mutation 113 D/R impairs binding [Moore (1994c)]

---

313 12G10 gp160(111–120) gp120(101–120 LAI) LWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- 12G10: The relative affinity for denatured/native gp120 is 17 – mutation 117 K/W impairs binding [Moore (1994c)]

---

314 135/9 (87–135/9) gp160(111–120) gp120(111–120 LAI) LWDQSLKPCV L Vaccine murine(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: C1 **Donor:** Matthias Niedrig
References: [Niedrig (1992b), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Trkola (1996a), Binley (1997a), Kropelin (1998)]

- 135/9: Defines the epitope as gp120(114–123) MHEDIISLWD (core LWD?) – weak neutralization of lab strain [Niedrig (1992b)]
- 135/9: The relative affinity for denatured/native gp120 is 15 – mutation 113 D/R impairs binding to native and denatured, 113 D/A only to denatured [Moore (1994c)]
- 135/9: Substitutions 106 E/A, 113 D/A or R, and 117 K/W impair binding, some substitutions enhance binding [Moore (1994d)]
- 135/9: Binding is enhanced by some anti-C1 and anti-C5 antibodies – enhances binding of some anti-V3, anti-C4 and anti-V2 MAbs – 135/9 binds to predicted alpha-helix in C1 [Moore & Sodroski(1996)]
- 135/9: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 135/9: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]
- 135/9: Noted to bind to C1 peptide HEDIISLWDQSLK – blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]

---

315 7C10 gp160(111–120) gp120(101–120 LAI) LWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- 7C10: The relative affinity for denatured/native gp120 is 5.8 – mutation 117 K/W impairs binding [Moore (1994c)]

---

316 C4 gp160(111–120) gp120(101–120 LAI) LWDQSLKPCV Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C1 **Donor:** George Lewis
References: [Abacioglu (1994), Moore & Ho(1993), Moore (1994c)]

- C4: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- C4: C1 region – epitope boundaries mapped by peptide scanning, BH10 core IISLW [Abacioglu (1994)]
- C4: The relative affinity for denatured/native gp120 is 10 [Moore (1994c)]

---

317 MF46.1 gp160(111–120) gp120(101–120 LAI) LWDQSLKPCV Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C1 **References:** [Thiriart (1989), Moore (1994c)]

- MF46.1: The relative affinity for denatured/native gp120 is 8.5 [Moore (1994c)]

---

318 6D5 gp160(122–141) gp120(122–141 LAI) LTPLCVSLKCTDLKNDTNTN Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: V2 **Donor:** S. Nigida and L. Arthur, NCI, Frederick, MD USA
References: [Moore (1994c), Moore (1994d)]

- 6D5: The relative affinity for denatured/native gp120 is 15 – mutations Δ119–205 and 125 L/G impair binding [Moore (1994c)]

---

319 B33 gp160(123–142) gp120(123–142 LAI) TPLCVSLKCTDLGNATNTNS no Vaccine murine(IgG2bκ)

Vaccine: *Vector/type:* recombinant protein *Strain:* NL43 *HIV component:* gp160

Ab type: V2 **Donor:** Daniels
References: [Abacioglu (1994), Bristow (1994)]

- B33: There are two MAbs in the literature named B33, see also gp160(727–734) [Abacioglu (1994)]
- B33: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]
- B27: C1 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)]
- B33: UK Medical Research Council AIDS reagent: ARP304, gp160/41 binding

---

320 polyclonal (VEI1) gp160(131–151) Env(131–151) CTDLKNDTNTNSSSGRMMME-K HIV-1 infection human( )

References: [Carlos (1999)]

- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

---

321 2H1B | gp160(155–161) | gp120(370–376 HIV2ROD) | RNISFKA | no | Vaccine | murine( )

Vaccine: *Vector/type:* peptide *Strain:* HIV-2 ROD

Ab type: C3 **References:** [Matsushita (1995)]

- 2H1B: Binds in WB, but binds poorly to Env on the cell surface [Matsushita (1995)]

---

322 697-D (697D, 697-30D, 697/30D) | gp160(dis 161–180) | gp120(dis 161–180 IIIB) | ISTSIRGKVQKEYAFFYKLD | P (weak) | HIV-1 infection | human(IgG1λ)

Ab type: V2 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center) or Cellular Products Inc, Buffalo NY

References: [Gorny (1994), Forthal (1995), Moore & Ho(1995), Trkola (1996a), Binley (1997a), Fouts (1997), Parren (1997b), Nyambi (1998), Stamatatos & Cheng-Mayer(1998), Gorny (2000), Hioe (2000), Nyambi (2000)]

- 697-D: Conformational with weak reactivity to V2 peptide ISTSIRGKVQKEYAFFYKLD – neutralized 3/4 primary isolates, but none of 4 lab strains – V2 substitutions 176/177 FY/AT, 179/180 LD/DL, 183/184 PI/SG, and 192–194 YSL/GSS abrogate binding – anti-C4 MAbs G3-536 and G45–60 enhance binding – mild oxidation of carbohydrate moieties inhibits binding [Gorny (1994)]
- 697-D: Not neutralizing, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 697-D: Review: called 697/30D – neutralizes some primary, but not lab adapted strains [Moore & Ho(1995)]
- 697-D: Partial inhibition of gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 697-D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 697-D bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)]
- 697-D: Does not neutralize TCLA strains but neutralizes some primary isolates weakly [Parren (1997b)]
- 697-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – V2 Abs 697-D, 1361, and 1357 tended to bind weakly with a similar pattern of specificity to virions, and bound well to soluble gp120: weak binding to 1/4 B clade viruses (CA5), and weak binding to viruses from subtype A and D [Nyambi (1998)]
- 697-D: Called 697-30D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)]
- 697-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V2 MAbs 697-D, 1357 and 1361 favored the monomer by approximately 2 fold[Gorny (2000)]

- 697-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V2 MAb 697-D did not effect proliferation [Hioe (2000)]
- 697-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 anti-V2 MAbs, which showed weak and sporadic binding, with the most frequent binding to C and D clades [Nyambi (2000)]

---

323 6C4/S | gp160(162–169) | gp120(BH10) | STSIRGKV | | Vaccine | ( )

Vaccine: *Vector/type:* protein *Strain:* BH10 *HIV component:* gp120

Donor: S. Ranjbar (NIBSC, UK)
References: [Moore (1993a)]

- 6C4/S: UK Medical Research Council AIDS reagent: ARP3049

---

324 C108G | gp160(162–169) | gp120(162–169 HXB2) | STSIRGKV | L | HIV-1 infection | chimpanzee(IgG1κ)

Donor: S. Tilley, Public Health Research Institute, NY, NY
References: [Warrier (1994), Wu (1995), Warrier (1995), Warrier (1996), Ugolini (1997), Mondor (1998), Alsmadi & Tilley(1998)]

- C108G: Chimps were infected with HIV-1 IIIB, and this high affinity MAb gave potent neutralization of HIV-1 IIIB – binding not affected by reduction of disulfide bonds – binding disrupted by removal of N-linked glycans – peptide binding lower affinity than glycosylated Env [Warrier (1994)]
- C108G: Strain specificity: LAI, Bal, HXB2 – conformational character – glycosylation site at 160 critical – mutation of conserved glycosylation site at 156 increased epitope exposure [Wu (1995)]
- C108G: Characterization of MAb variable region [Warrier (1995)]
- C108G: Synergistic neutralization of HIV-1 when combined with anti-V3 MAbs 0.5β and C311E, or anti-CD4BS MAbs, 1125H and 5145A – neutralization further enhanced by presence of both 1125H and 0.5β [Warrier (1996)]
- C108G: Viral binding inhibition by C108G was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- C108G: Inhibits HX10 binding to both CD4 positive and negative HeLa cells[Mondor (1998)]
- C108G: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against only IIIB – this is first demonstration of ADCC directed by a V2 specific MAb [Alsmadi & Tilley(1998)]

---

325 10/76b | gp160(162–170) | gp120(162–171 BH10) | STSIRGKVQ | L (HXB10) | Vaccine | rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [McKeating (1993b), McKeating (1993a), Shotton (1995), Wu (1995), McKeating (1996)]

- 10/76b: R to L substitution abrogated binding – human sera recognize epitope [McKeating (1993b)]
- 10/76b: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)]

- 10/76b: Included in cross-competition and neutralization studies [Shotton (1995)]
- 10/76b: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)]
- 10/76b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 10/76b: UK Medical Research Council AIDS reagent: ARP3077

---

326 11/41e gp160(162–170) gp120(162–171) STSIRGKVQ L (HXB10) Vaccine rat(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [McKeating (1993b), Shotton (1995), Wu (1995)]

- 11/41e: R to L abrogated binding – human sera recognize the epitope [McKeating (1993b)]
- 11/41e: Included in cross-competition and neutralization studies [Shotton (1995)]
- 11/41e: HX10 strain specificity – binds native and deglycosylated gp120 [Wu (1995)]

---

327 11/4b gp160(162–170) gp120(162–171) STSIRGKVQ L (HXB10) Vaccine rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [McKeating (1993b), Shotton (1995), Wu (1995), Moore & Sodroski(1996)]

- 11/4b: A mutation R166L abrogated binding – human sera recognize epitope [McKeating (1993b)]
- 11/4b: Cross-competes with MAbs 10/76b and 11/4c – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)]
- 11/4b: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)]
- 11/4b: Linear V2 epitope – reciprocal binding enhancement of anti-V2 discontinuous epitope antibodies (in contrast to BAT085) and CD4 inducible antibody 48d. Reciprocal inhibits BAT085 binding – inhibits CRA-3 binding CRA-3 does not inhibit 11/4b [Moore & Sodroski(1996)]

---

328 RSD-33 gp160(162–170) gp120(162–171) STSIRGKVQ Vaccine ( )

Vaccine: *Vector/type:* protein *Strain:* BH10 *HIV component:* gp120

Donor: R. Daniels (NIMR, UK)
References: [Moore (1993a)]

---

329 11/4c (11/4c/1j/4j) gp160(162–170) gp120(152–181) STSIRGKVQ L (HXB2) Vaccine rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V2 **References:** [McKeating (1993b), Wu (1995), Shotton (1995), Peet (1998)]

- 11/4c: R to L substitution abrogated binding – human sera recognize epitope [McKeating (1993b)]
- 11/4c: HX10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)]
- 11/4c: Cross-competes with MAbs 10/76b and 11/4b – HXB2 neutralization escape mutant has the substitution I/T at residue 165 [Shotton (1995)]

- 11/4c: Called 11/4c/1j/4j – The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – 11/4c was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 11/4c: UK Medical Research Council AIDS reagent: ARP3035

---

330 12b gp160(162–181) gp120(162–181) STSIRGKVQKEYAFFYKLDI L (HXB10) Vaccine rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V2 **References:** [Shotton (1995), McKeating (1996)]

- 12b: V2 MAb neutralized HXB2 – position 179–180 LD to DL abrogates binding – competes with 60b, but not 74 [Shotton (1995)]
- 12b: Neutralizes HXB2, but fails to neutralize chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]

---

331 G3-136 (G3.136) gp160(dis 170–180) gp120(dis 170–180 IIIB) QKEYAFFYKLD L Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: V2 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Fung (1992), Pirofski (1993), Thali (1993), Moore & Ho(1993), Moore (1993a), Yoshiyama (1994), Sattentau & Moore(1995), Stamatatos & Cheng-Mayer(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000)]

- G3-136: V2 region – binds and neutralizes IIIB and RF in CEM-SS cells, but not MN – neutralization activity against a few primary isolates in PBMC – sCD4 binding inhibits binding (contrast with BAT085) – deglycosylation or reduction of gp120 by DTT diminishes reactivity [Fung (1992)]
- G3-136: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-136: Marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore (1993a)]
- G3-136: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore (1993a)]
- G3-136: HIV-1 RF V2 substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity [Yoshiyama (1994)]
- G3-136: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V2 loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a – anti-V2 MAbs G3-4 and G3.136 don‘t bind to T-cell tropic SF2 [Stamatatos & Cheng-Mayer(1995)]
- G3-136: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)]

- G3-136: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50–69, in contrast to anti-V3 MAbs [Poignard (1996a)]
- G3-136: Called G3.136 – does not mediate gp120 virion dissociation in contrast to anti-V2 MAb G3-4 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos (1997)]
- G3-136: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-136: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-136: Called G3.136 – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)]
- G3-136: Called G3.136 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

---

332 G3-4 (G3.4) | gp160(dis 170–180) | gp120(dis 170–180 BH10) | QKEYAFFYKLD | L | Vaccine | murine(IgG2bκ)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V2 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Ho (1991a), Ho (1992), Fung (1992), McKeating (1992a), Moore & Ho(1993), Sullivan (1993), Sattentau (1993), Thali (1993), Moore (1993a), Moore (1994b), Gorny (1994), Thali (1994), Yoshiyama (1994), Stamatatos & Cheng-Mayer(1995), Wu (1995), Sattentau & Moore(1995), Jagodzinski (1996), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Stamatatos (1997), Ditzel (1997), Wyatt (1997), Parren (1998a), Stamatatos & Cheng-Mayer(1998), Ly & Stamatatos(2000), Srivastava (2002)]

- G3-4: Binding is sensitive to removal of glycans by endo H – 50% neutralization of 4/9 primary isolates – has conformational features [Ho (1991a)]
- G3-4: Neutralizes IIIB and RF, not MN – blocks sCD4-gp120, not as potent as MAb 15e – V2 binding MAbs BAT085 and G3-136 block G3-4 gp120 binding – sensitive to reduction of gp120 by DTT [Ho (1992)]
- G3-4: Substitutions in residues 176 to 184 affect MAb recognition – substitutions in V2 can result in gp120-gp41 dissociation [Sullivan (1993)]
- G3-4: Increased binding in the presence of sCD4 [Sattentau (1993)]
- G3-4: Conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- G3-4: V2 region, marginal binding to peptide, binding inhibited by 183/184 PI/SG substitution [Moore (1993a)]

- G3-4: Conformationally sensitive – sporadic cross-reactivity among, and outside, B clade gp120s [Moore (1994b)]
- G3-4: Weakly neutralizing, IC 50 = 53 μg/ml [Gorny (1994)]
- G3-4: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter G3-4s ability to neutralize [Thali (1994)]
- G3-4: Neutralizes RF – substitutions 177 Y/H and 179 L/P in the V2 loop of RF reduce affinity and result in neutralization escape [Yoshiyama (1994)]
- G3-4: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V2 loop epitopes are somewhat occluded in the oligomeric gp120 epitopes on the virion surface relative to the gp120 monomer as indicated by an increase in the half-maximal binding values to macrophage-tropic isolates SF162 and SF128a – anti-V2 MAbs G3-4 and G3.136 don't bind to T-cell tropic SF2 [Stamatatos & Cheng-Mayer(1995)]
- G3-4: Reactive with BH10, RF, and MN – binds native, but not denatured or deglycosylated gp120, binds to deglycosylated V1V2 fusion protein, suggesting importance of glycans outside the V1V2 region [Wu (1995)]
- G3-4: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes Hx10 cell-free virus [Sattentau & Moore(1995)]
- G3-4: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – deletion of the V3 loop from gp120 results in more potent G3-4 binding inhibition by CRDS – G3-4 epitope described as 176–184 FYKLDIIPI and 191–193 YSL [Jagodzinski (1996)]
- G3-4: Binding enhanced by selected antibodies to C1, C4, C5, V3 and anti-CD4 binding site MAbs – enhances binding of selected V3, C4 and anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-4: Described epitope as STSIRGKVKEYAFFYKLDI – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT085 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50–69, in contrast to anti-V3 MAbs [Poignard (1996a)]
- G3-4: Called G3.4 – mediates gp120 virion dissociation in contrast to anti-V2 MAb G3-136 – not neutralizing for SF162 or SF128A in either primary macrophages or PBMC [Stamatatos (1997)]
- G3-4: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-4: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-4: Called G3.4 – Deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V2 MAbs G3.4, G3.136, or 687–30D [Stamatatos & Cheng-Mayer(1998)]
- G3-4: Called G3.4 – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

- G3-4: Called G3.4 – Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – G3.4 recognized o-gp140 [Srivastava (2002)]

---

333 BAT085 (BAT-085) | gp160(171–180) | gp120(170–180 IIIB) | KEYAFFYKLD | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

Donor: Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung (1987), Fung (1992), Moore & Ho(1993), Pirofski (1993), Thali (1993), Moore (1993a), D'Souza (1994), Moore (1994d), Gorny (1994), Yoshiyama (1994), Wu (1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Ditzel (1997), Parren (1998a)]

- BAT085: V2 region – sCD4 does not block – neutralizes IIIB and some primary isolates, but not MN or RF – binds MN – deglycosylation or DDT reduction of gp120 does not diminish reactivity [Fung (1992)]
- BAT085: Called BAT-85 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120, and does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT085: 7/8 V2 murine MAbs required gp120 native structure to bind, but BAT085 was the exception – type-specific [Moore (1993a)]
- BAT085: Peptide affinities of G3-136 and G3-4 are 100-fold less than BAT085, but BAT085 has lower affinity for BH10 gp120 and is weaker at neutralization [Moore (1993a)]
- BAT085: Multi-lab study for antibody characterization and assay comparison – did not bind MN or SF2 [D'Souza (1994)]
- BAT085: Interacts with two overlapping peptides with region of overlap KEYAFFYKLD [Gorny (1994)]
- BAT085: Neutralizes RF – substitution 177 Y/H in the V2 loop of RF does not inhibit neutralization, in contrast to MAbs G3-4 and SC258 [Yoshiyama (1994)]
- BAT085: HXB10 strain specificity – binds native, deglycosylated, or denatured gp120 [Wu (1995)]
- BAT085: Bound preferentially to the monomeric rather than oligomeric form of LAI gp120 – neutralizes cell free Hx10 [Sattentau & Moore(1995)]
- BAT085: Binding is blocked by other V2 region antibodies, enhanced by several anti-C1 MAbs, and anti-V3 MAb G511 – reciprocal enhancement of CD4i MAb 48d binding [Moore & Sodroski(1996)]
- BAT085: Epitope suggested to be QKEYAFFYKLD – binds oligomer – binding of V2 MAbs G3-136, G3-4 or BAT123 did not significantly alter gp120 dissociation from virus or expose the gp41 epitope of MAb 50–69, in contrast to anti-V3 MAbs [Poignard (1996a)]
- BAT085: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

---

334 60b | gp160(172–181) | gp120(172–181 HXB2) | EYAFFYKLDI | no | Vaccine | rat(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [Shotton (1995)]

- 60b: V2 MAb did not neutralize HXB2 – bound to rgp120 in ELISA – substitutions 179–180 LD/DL and 191–193 YSL/GSS abrogate binding, as do changes outside the minimum epitope – competes with 12b, but not 74 [Shotton (1995)]

---

335 74 gp160(172–181) gp120(172–181) EYAFFYKLDI no Vaccine rat(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

References: [Shotton (1995)]

- 74: V2 MAb did not neutralize HXB2 – did not bind rgp120 ELISA – position 179–180 LD to DL abrogates binding, as do changes outside the minimum epitope – does not compete with 60b or 12b, and is enhanced by two conformation dependent MAbs [Shotton (1995)]

---

336 38/12b gp160(172–191) gp120(172–191 HXB2) EYAFFYKLDIIPIDNDTTSY Vaccine rat( )

Vaccine: *Vector/type:* protein *Strain:* BH10 *HIV component:* gp120

References: [Wu (1995)]

- 38/12b: Broad specificity: HXB2, MN, SF162 – binds native and deglycosylated gp120 [Wu (1995)]

---

337 38/60b gp160(172–191) gp120(172–191 HXB2) EYAFFYKLDIIPIDNDTTSY Vaccine rat( )

Vaccine: *Vector/type:* protein *Strain:* BH10 *HIV component:* gp120

References: [Wu (1995)]

- 38/60b: Strain specificity: HXB2 – binds native and deglycosylated gp120 [Wu (1995)]

---

338 polyclonal (VEI2) gp160(176–196) Env( ) FYKLDIVPIDNTTTSYRLISC HIV-1 infection human( )

References: [Carlos (1999)]

- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

---

339 322–151 gp160(211–221) gp120(201–220 LAI) EPIPIHYCAPA Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Donor: G. Robey, Abbot Labs
References: [Moore (1994c), Moore (1994d)]

- 322–151: The relative affinity denatured/native gp120 is 30 [Moore (1994c)]

---

340 3D3.B8 gp160(211–221) gp120(211–220 LAI) EPIPIHYCAPA Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

References: [Bolmstedt (1990), Moore (1994c)]
- 3D3.B8: The relative affinity denatured/native gp120 is greater than 10 [Moore (1994c)]

---

341 4C11.D8 gp160(211–221) gp120(211–220 LAI) EPIPIHYCAPA Vaccine murine(IgM)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

References: [Bolmstedt (1990), Moore (1994c)]
- 4C11.D8: The relative affinity denatured/native gp120 is greater than 10 [Moore (1994c)]

---

342 493–156 gp160(211–230) gp120(211–230 LAI) EPIPIHYCAPAGFAILKCNN Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Donor: G. Robey, Abbot Labs
References: [Moore (1994c)]
- 493–156: The relative affinity denatured/native gp120 is >10 [Moore (1994c)]

---

343 110.1 gp160(212–221) gp120(200–217) PIPIHYCAPA no Vaccine human( )

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

References: [Pincus & McClure(1993), Pincus (1996), Valenzuela (1998)]
- 110.1: There is another antibody with this ID that binds to Env at positions 491–500 in LAI, see [Gosting (1987)]
- 110.1: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding – 110.1-RAC did not mediate cell killing, and sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]

---

344 GV4H3 gp160(219–226) gp120(219–226 IIIB) APAGFAIL Vaccine murine( )

Vaccine: *Vector/type:* protein-Ab complex *HIV component:* gp120 complexed with MAb M77

References: [Denisova (1996)]
- GV4H3: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes [Denisova (1996)]

---

345 J1 gp160(222–231) gp120(222–231 LAI) GFAILKCNNK Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* LAI

Donor: J. Hoxie, U. Penn.
References: [Moore (1994c), Moore (1994d), Cook (1994)]
- J1: The relative affinity denatured/native gp120 is 30 [Moore (1994c)]
- J1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]

---

346 J3 gp160(222–231) gp120(222–231 LAI) GFAILKCNNK Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* LAI

Donor: J. Hoxie, U. Penn.
References: [Moore (1994c), Cook (1994)]

- J3: The relative affinity denatured/native gp120 is 30 [Moore (1994c)]
- J3: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the N-terminal half of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]

---

347 1006–30-D gp160(236–245) gp120(241–251) KGSCKNVSTV human(IgG1λ)
Ab type: C2 **References:** [Hioe (2000), Nyambi (2000)]

- 1006–30-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C2 MAbs 1006–30-D and 847-D did not effect proliferation [Hioe (2000)]
- 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MABs was weak to isolates from clades B, C, D, E, F, G, and they did not not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)]

---

348 847-D gp160(236–245) gp120(241–251) KGSCKNVSTV human(IgG1λ)
Ab type: C2 **References:** [Hioe (2000), Nyambi (2000)]

- 847-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C2 MAbs 1006–30-D and 847-D did not effect proliferation [Hioe (2000)]
- 847-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including two C2 MAbs – the binding of anti-C2 MABs was weak to isolates from clades B, C, D, E, F, G, and they did not not bind to isolates from subtypes A and H – epitope is suggested to be in a 20 amino acid peptide KGSCKNVSTVQCTHGIRPVV [Nyambi (2000)]

---

349 MF169.1 gp160(252–261) gp120(242–261 LAI) RPVVSTQLLL Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

References: [Thiriart (1989), Moore (1994c), Moore (1994d)]

- MF169.1: The relative affinity denatured/native gp120 is 11 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)]

---

350 MF170.1 gp160(252–261) gp120(242–261 LAI) RPVVSTQLLL Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

References: [Thiriart (1989), Moore (1994c), Moore (1994d)]

- MF170.1: The relative affinity denatured/native gp120 is 15 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding to denatured and native gp120, and 262N/T, 269 E/L and 281 A/V to only native gp120 [Moore (1994c)]

---

351 MF87.1 gp160(252–261) gp120(242–261 LAI) RPVVSTQLLL Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

References: [Thiriart (1989), Moore (1994c)]

- MF87.1: The relative affinity denatured/native gp120 is 10 – mutations 252 R/W, 257 T/G, and 257 T/R impair binding [Moore (1994c)]

---

352 213.1 gp160(252–261) gp120(242–261 LAI) RPVVSTQLLL Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *HIV component:* Env

Ab type: C2 **Donor:** Claudine Bruck
References: [Thiriart (1989), Moore & Ho(1993), Moore (1994c)]

- 213.1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)]
- 213.1: The relative affinity denatured/native gp120 is 100 – mutations 252 R/W, 257 T/G or T/R impair binding [Moore (1994c)]
- 213.1: UK Medical Research Council AIDS reagent: ARP334

---

353 B12 gp160(252–271) gp120(252–271 LAI) RPVVSTQLLLNGSLAEEEVV Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Moore (1994c)]

- B12: C2 region – the relative affinity for denatured/native gp120 is 27 – mutations 257 T/R and 262 N/T impair binding [Moore (1994c)]

---

354 B13 (Bh13) gp160(252–271) gp120(252–271 LAI) RPVVSTQLLLNGSLAEEEVV Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **Donor:** George Lewis, Institute of Human Virology, Baltimore MD, USA
References: [Pincus & McClure(1993), Moore & Ho(1993), Moore (1994c), Abacioglu (1994), Moore (1994d), Pincus (1996), Connor (1998)]

- B13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- B13: The relative affinity for denatured/native gp120 is 30 – mutations 257 T/R and 269 E/L impair binding [Moore (1994c)]
- B13: C2 region – epitope boundaries mapped by peptide scanning, core epitope: TQLLLN [Abacioglu (1994)]
- B13: Called Bh13 – binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]

---

355 C13 gp160(252–271) gp120(252–271 LAI) RPVVSTQLLLNGSLAEEEVV Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **Donor:** George Lewis
References: [Moore & Ho(1993), Moore (1994c), Abacioglu (1994)]

- C13: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- C13: The relative affinity for denatured/native gp120 is 36 – mutations 257 T/R, 267 E/L, and 269 E/L impair binding [Moore (1994c)]

- C13: Epitope boundary extended to RPVVSTQLLLNGSLAEEEVVIR, to take into account the effect of a point mutation [Abacioglu (1994)]
- C13: NIH AIDS Research and Reference Reagent Program: 1209

---

356 M89 gp160(252–271) gp120(252–271 LAI) RPVVSTQLLLNGSLAEEEVV no Vaccine murine(IgG1)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: C2 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d)]

- M89: Immunoblot reactive, RIP negative, for strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]
- M89: C2 region – the relative affinity for denatured/native gp120 is >30 – mutations 257 T/R and 269 E/L impair binding [Moore (1994c)]

---

357 B21 gp160(257–262) gp120(257–262 BH10) TQLLLN Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B21: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

358 B23 gp160(257–262) gp120(257–262 BH10) TQLLLN Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B23: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

359 B24 gp160(257–262) gp120(257–262 BH10) TQLLLN Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B24: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

360 B25 gp160(257–262) gp120(257–262 BH10) TQLLLN Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B25: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

361	B3	gp160(257–262)	gp120(257–262 BH10)	TQLLLN	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B3: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

362	B26	gp160(257–263)	gp120(257–263 BH10)	TQLLLNG	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B26: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

363	B29	gp160(257–263)	gp120(257–263 BH10)	TQLLLNG	Vaccine	murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B29: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

364	B36	gp160(257–263)	gp120(257–263 BH10)	TQLLLNG	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C2 **References:** [Abacioglu (1994)]

- B36: C2 region, epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

365	110.E	gp160(262–281)	gp120(262–281 LAI)	NGSLAEEEVVIRSVNFTDNA	Vaccine	murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C2 **Donor:** F. Traincard
References: [Moore (1994c), Moore (1994d)]

- 110.E: The relative affinity for denatured/native gp120 is 7.3 [Moore (1994c)]

366	110.C	gp160(271–280)	gp120(271–280 LAI)	VIRSVNFTDN	Vaccine	murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C2 **Donor:** F. Traincard, Hybridolabs, Institut Pasteur
References: [Moore (1994c), Moore (1994d), Valenzuela (1998)]

- 110.C: The relative affinity for denatured/native gp120 is 1 [Moore (1994c)]
- 110.C: Only slightly reduces LAI viral binding or entry into CEM cells [Valenzuela (1998)]

367	IIIB-V3–26	gp160(291–307)	gp120(299–304 IIIB)	SVEINCTRPNNNTRKSI	no	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB

Ab type: V3 **References:** [Laman (1992)]

- IIIB-V3–26: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)]

368	IIIB-V3–21 (V3–21)	gp160(294–299)	gp120(299–304 IIIB)	INCTRP	no	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB

Ab type: V3 **Donor:** J. Laman
References: [Laman (1992), Laman (1993), Valenzuela (1998)]

- IIIB-V3–21: Binds to the base of the V3 loop on denatured gp120 [Laman (1992)]
- IIIB-V3–21: Binds to NP40 treated gp120, and epitope is probably obscured by local glycosylation [Laman (1993)]
- IIIB-V3–21: Does not block HIV-1 LAI binding or entry into CEM cells [Valenzuela (1998)]
- IIIB-V3–21: UK Medical Research Council AIDS reagent: ARP3048
- IIIB-V3–21: NIH AIDS Research and Reference Reagent Program: 1725

369	polyclonal	gp160(296–327)	gp120(MN)	CNYNKRKRIHIGPGRAFYTTK-NIIGTIC	L		rabbit(IgA,IgG)

Ab type: V3 **References:** [FitzGerald (1998)]

- Polyclonal response to MN, or Thai E V3 loop inserted into Pseudomonas Exotoxin for vaccination – inserts of 14 or 26 amino acids were used from MN or a Thai E strain, constrained by disulfide bond – sera from vaccinated rabbit were reactive with strain-specific gp120 – administration to mucosal surfaces elicits IgA [FitzGerald (1998)]

370	polyclonal	gp160(297–320)	gp120( )	NYNKRKRIHIGPGRAFYTTK	L	HIV-1 infection, Vaccine	human( )

Vaccine: *Vector/type:* peptide *Strain:* cocktail *HIV component:* V3

Ab type: V3 **References:** [Bartlett (1998)]

- V3 peptide vaccine (MN, RF, EV91, and Can0A) with a C4 helper T cell epitope were used to vaccinate HLA-B7 HIV-infected patients – V3 Ab levels and the anti-HIV proliferative response, but no decrease in HIV-1 RNA levels or increase in CD4 levels was observed [Bartlett (1998)]

371	polyclonal	gp160(297–320)	gp120( )	NYNKRKRIHIGPGRAFYTTK		HIV-1 exposed seronegative	human(IgA)

Ab type: V3 **References:** [Kaul (1999)]

- HIV-1 Env-specific mucosal IgA found in genital track of 16/21 HIV-1 resistant chronically exposed Kenyan sex workers – 11/21 had detectable Th responses [Kaul (1999)]

372	polyclonal	gp160(297–331)	Env(303–335 LAI)	TRPNNNTRKSIHIGPGRAFYA-TGEIIGDIRQAH	no	Vaccine	human(IgG)

Vaccine: *Vector/type:* lipopeptide *Strain:* LAI *HIV component:* V3 *Stimulatory Agents:* QS21

Ab type: V3 **References:** [Pialoux (2001)]

- 28 subjects were vaccinated with six HIV-1 peptides that were selected to be particularly rich in CTL epitopes, presented in lipopeptides with or without adjuvant QS21 – HIV-specific Ab responses were detected in 25/28 (89%), proliferative in 19/28 (79%), and CTL in 13/24 (54%) of testable volunteers – 14/28 had non-neutralizing Ab responses to this peptide (E), 7/24 had proliferative responses, and multiple CTL responses were detected [Pialoux (2001)]

373	MO97/V3	gp160(299–308)	gp120(299–308 IIIB)	PNNNTRKSIR	no	*in vitro* stimulation	human(IgM)

Ab type: V3 **References:** [Ohlin (1992)]

- MO97: Generated through *in vitro* stimulation of uninfected-donor lymphocytes with rpB1 (IIIB Env 286–467) [Ohlin (1992)]

374	polyclonal	gp160(299–331)	gp120(306–338 BH10)	PNNNTRKSIRIQRGPGRAFVT-IGKIGNMRQAHC	L	Vaccine	rabbit(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10

Ab type: V3 **References:** [Neurath & Strick(1990)]

- 21 V3 loop variant peptides spanning this region were tested and serological cross-reactivity correlated with divergence [Neurath & Strick(1990)]

375	55/1l	gp160(300–315)	gp120(300–315)	NNNTRKRIRIQRGPGR?			( )

Ab type: V3 **References:** [Peet (1998)]

- 55/1l: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 55/1l binding was abrogated by V3 serine substitutions in the V3 loop – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

376	8/38c (8/38/1c)	gp160(300–315)	gp120(300–315 HXB10)	NNNTRKRIRIQRGPGR	L	Vaccine	rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **Donor:** C. Dean and C. Shotton, Institute for Cancer Research, Surrey, UK
References: [McKeating (1992a), Sattentau & Moore(1995), Jeffs (1996), Parren (1998a), Peet (1998)]

- 8/38c: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating (1992a)]
- 8/38c: Binds equally well to monomer and oligomer, less rapid association rate than other anti-V3 antibodies, and an associated less potent neutralization of lab strains [Sattentau & Moore(1995)]
- 8/38c: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 8/38c: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

- 8/38c: Called 8/38/1c: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 8/38c binding was diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 8/38c: UK Medical Research Council AIDS reagent: ARP3039

---

377 8/64b | gp160(300–315) | gp120(300–315 HXB10) | NNNTRKRIRIQRGPGR | L | Vaccine | rat(IgM)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), Peet (1998)]

- 8/64b: Binds to virion gp120 and neutralizes only in the presence of sCD4 [McKeating (1992a)]
- 8/64b: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, and anti-V3 MAb 8/64b binding was abrogated by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- 8/64b: UK Medical Research Council AIDS reagent: ARP3036

---

378 polyclonal | gp160(300–322) | gp120(IIIB) | CNNTRKSIRIQRGPGRAFVTI-GK | L | | guinea pig(IgG)

Ab type: V3 **Donor:** D. Bolognesi and T. Matthews, Duke University
References: [Allaway (1993)]

- Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]

---

379 polyclonal | gp160(300–328) | Env( ) | NNNTRKSIRIGPGRAFYTTGD-IGNIRQ | | HIV-1 infection | human( )

Ab type: V3 **References:** [Carlos (1999)]

- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

---

380 9284 (NEA 9284) | gp160(301–312) | gp120(307–318 IIIB) | NNTRKSIRIQRG | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

Ab type: V3 **Donor:** Dupont de Nemours, Les Ulis, France or Wilmington, Delaware
References: [Skinner (1988b), Skinner (1988a), Sattentau & Moore(1991), Wyatt (1992), McKeating (1992a), Sattentau (1993), Moore (1993b), Trujillo (1993), Thali (1993), VanCott (1994), Thali (1994), Cook (1994), Okada (1994), Sorensen (1994), Sattentau & Moore(1995), VanCott (1995), Fontenot (1995), Moore & Sodroski(1996), Poignard (1996a), Cao (1997), Binley (1997a), Parren (1998a), Schonning (1998)]

- 9284: IIIB type-specific binding and neutralization [Skinner (1988b)]
- 9284: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 9284: Single amino acid substitutions in the C4 region (427 W/V or W/S) or at the base of the V3 loop (298 R/G) can significantly increase binding and neutralization– position 427 is also important for CD4 binding and anti-CD4 binding site MAbs [Wyatt (1992)]
- 9284: Increased binding in the presence of sCD4 [Sattentau (1993)]
- 9284: Inhibits C4 region antibodies (G3-299, G3-519) which have conformational requirements [Moore (1993b)]
- 9284: Peptide RIQRGPGRAFVTIGKIGNMRQA – Reacts with three human brain proteins of 35, 55, 110 kd – called NEA-9284 [Trujillo (1993)]
- 9284: Does not bind MN gp120, just IIIB [VanCott (1994)]
- 9284: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 9284: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]
- 9284: Binding domain aa 301–310: TRKSIRIQRG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β – called NEA9284 [Okada (1994)]
- 9284: Did not neutralize infection of HIV/HTLV-I pseudotype [Sorensen (1994)]
- 9284: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free virus Hx10 [Sattentau & Moore(1995)]
- 9284: Used to monitor HIV-1 Env expression in infected H9 cells, binds native and reduced gp120s similarly [VanCott (1995)]
- 9284: Binds V3 loop – anti-C1 MAbs 133/290 and 135/9 enhance binding – reciprocal binding inhibition of other anti-V3 MAbs [Moore & Sodroski(1996)]
- 9284: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- 9284: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao (1997)]
- 9284: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 9284: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, 9284 was found to have an inaccessible epitope on the oligomeric form of Env and anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]

381	polyclonal	gp160(301–325)	gp120(IIIB)	NNTRKSIRIQRGPGRAFVTIG-KIGN	L	Vaccine	murine(IgA)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *Stimulatory Agents:* cholera toxin adjuvant

Ab type: V3 **References:** [Bukawa (1995)]

- Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa (1995)]

---

382 polyclonal | gp160(301–325) | gp120(IIIB) | NNTRKSIRIQRGPGRAFVTIG-KIGN | L | Vaccine | murine(IgA22a)

Vaccine: *Vector/type:* DNA *Strain:* IIIB *HIV component:* Env, Rev *Stimulatory Agents:* QS-21

Ab type: V3 **References:** [Sasaki (1998)]

- An anti-Env response was sought, and co-expression of Rev was required – intramuscular versus nasal vaccination with DNA vaccine with a QS-21 adjuvant was studied – QS-21 enhanced the IgG2a response mediated via Th1 cytokines IFNgamma and IL-2 [Sasaki (1998)]

---

383 polyclonal | gp160(302–318) | Env( ) | NTRKSIHIGPGRAFY | L P | HIV-1 infection | human( )

Ab type: V3 **References:** [Bongertz (2001)]

- Non-transmitting mothers had an increased frequency of high neutralizing plasma Ab titers against HIV-1 MN (1:50 dilution, >90% neutralization, 33/88 pregnant women), compared to plasma from transmitting mothers (0/8 pregnant women) – non-transmitting mothers also had more potent neutralization against primary isolates from transmitting mothers, but neutralization of autologous virus was comparable for non-transmitting (7/13) and transmitting mothers (2/4) [Bongertz (2001)]

---

384 MAG 109 | gp160(302–321) | gp120(302–321 BH10) | NTRKSIRIQRGPGRAFVTIG | L | Vaccine | murine( )

Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994)]

- MAG 109: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang (1994)]

---

385 MAG 49 (#49) | gp160(302–321) | gp120(302–321 BH10) | NTRKSIRIQRGPGRAFVTIG | L | Vaccine | murine( )

Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994), Moore & Sodroski(1996)]

- MAG 49: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang (1994)]
- MAG 49: Called #49 in this text. Binding enhanced by anti-C1 MAbs 133/290, 135/9, and by many anti-CD4 binding site MAbs – reciprocal enhancement of some anti-V2 MAbs – reciprocal binding inhibition of anti-V3 MAbs [Moore & Sodroski(1996)]

---

386 MAG 53 | gp160(302–321) | gp120(302–321 BH10) | NTRKSIRIQRGPGRAFVTIG | L | Vaccine | murine( )

Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994)]

- MAG 53: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang (1994)]

---

387 MAG 56 gp160(302–321) gp120(302–321) NTRKSIRIQRGPGRAFVTIG L Vaccine murine( )

Vaccine: *Vector/type:* sCD4-gp120 complex *Strain:* HXB2 *HIV component:* gp120

Ab type: V3 **References:** [Kang (1994)]

- MAG 56: Binds a V3 loop peptide – sensitive to both V3 loop mutations and a mutation at the base of the V1/V2 loop structure (120/121 VK/LE) [Kang (1994)]

---

388 1324-E (1324E) gp160(303–308) Env(Clade E) TRTSVR L HIV-1 infection human(IgG1κ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1324-E: A human MAb was derived from an HIV-1 E clade infection from a US service man who had served in Thailand, selected with the consensus V3 peptide from clade E – cross-reactive with V3 peptides, and gp120 from E, C and A clades, as well as cells infected with a C-clade primary isolate, but not with B and D clade V3 peptides or rgp120 – neutralizes E clade virus adapted for growth in H9 cells, but not 5 primary E clade isolates, including the autologous isolate – kinetic parameters were measured, 1324E was comparable to 447-52D [Gorny (1998)]
- 1324-E: E clade stimulated MAb did not cross-react with B clade peptides nor did B clade derived peptides with an E clade V3 loop, but both E and B clade stimulated Abs can cross-react with some peptides from other clades – this Ab showed strong binding to several E, A and F peptides, one C peptide, and no reactivity with B peptides and most D peptides [Zolla-Pazner (1999a)]
- 1324-E: MAb reacted with peptides from E clade, while B clade derived MAbs could not [Zolla-Pazner (1999b)]
- 1324-E: Called 1324E – A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1324E showed poor cross-reactivity, and was the only MAb tested that was derived from a non-B clade infected patient, an E clade infection was the source of 1324E [Nyambi (2000)]

---

389 polyclonal gp160(303–319) gp120(subtype C) CKRKIHIGPGQAFYT Vaccine murine(IgG2a,IgG2b)

Vaccine: *Vector/type:* peptide in ISCOM or liposome *HIV component:* V3 *Stimulatory Agents:* ISCOM

Ab type: V3 **References:** [Ahluwalia (1997)]

- A V3 loop peptide modified to resemble an Indian form (GPGQ) was incorporated into ISCOMS (immune stimulating complexes) or liposomes, and used to immunize mice – the IgG2a/IgG2b antibody response was enhanced by the presentation in the ISCOM suggestive of a Th1 response [Ahluwalia (1997)]

---

390 MO99/V3 gp160(304–308) gp120(304–308 IIIB) RKSIR no *in vitro* stimulation human(IgM)

Ab type: V3 **References:** [Ohlin (1992)]

- MO99: Generated through *in vitro* stimulation of uninfected-donor lymphocytes with rpB1 (IIIB Env 286–467) [Ohlin (1992)]

---

391 C311E gp160(304–313) gp120(309–316 MN) RKRIHIGP L HIV-1 infection chimpanzee(IgG1)
Ab type: V3 **References:** [Warrier (1996), Alsmadi & Tilley(1998)]
- C311E: Chimps were infected with HIV-1 IIIB, and this resulting MAb gave synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier (1996)]
- C311E: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – C311E bound and directed lysis against all four strains [Alsmadi & Tilley(1998)]

---

392 907 gp160(304–314) gp120(309–318) RKSIRIQRGPG L Vaccine murine(IgG1κ)
Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* gp160
References: [Chesebro & Wehrly(1988), Pincus (1989), Pincus (1991), Pincus (1996)]
- 907: Strain specific binding, and neutralization of only the LAV strain [Chesebro & Wehrly(1988)]
- 907: Coupled to ricin A chain (RAC), MAb 907 inhibited protein synthesis and cell growth in HIV-infected cells [Pincus (1989)]
- 907: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus (1991)]
- 907: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]

---

393 924 gp160(304–314) gp120(309–318 IIIB) RKSIRIQRGPG Vaccine murine(IgG1κ)
Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* gp160
Ab type: V3 **References:** [Chesebro & Wehrly(1988), Pincus (1991), Pincus & McClure(1993), Pincus (1993), Cook (1994), Pincus (1996), Pincus (1998)]
- 924: HIV IIIB strain specific [Chesebro & Wehrly(1988)]
- 924: Epitope sequence is based on database count of a specified location – 924-RAC immunotoxin is IIIB strain-specific [Pincus (1991)]
- 924: MAb was coupled to ricin A chain (RAC) – immunotoxin efficacy was not significantly decreased by sCD4, although the efficacy of gp41 MAb immunotoxins *in vitro* increased 30-fold by sCD4 [Pincus & McClure(1993)]
- 924: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – MAb 924 was used as a control – infected lab workers and a vaccinia gp160 vaccine had strong V3 MAb response, but alum absorbed rec gp160 did not generate anti-V3 response [Pincus (1993)]
- 924: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]
- 924: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]

---

394 polyclonal gp160(304–318) gp120(304–318 LAI) RKSIRIQRGPGRAFV *in vitro* stimulation human(IgG,IgM)
Ab type: V3 **References:** [Chin (1995)]
- Mimicking the humoral immune response *in vitro* supports isotype switching – human IgG MAbs were generated from naive donors [Chin (1995)]

---

395 polyclonal gp160(304–318) gp120(304–318 LAI) RKSIRIQRGPGRAFV Vaccine human(IgG,IgM)

Vaccine: *Vector/type:* peptide *Strain:* LAI

Ab type: V3 **References:** [Zafiropoulos (1997)]

- IgG to IgM isotype switching in response to primary and secondary peptide vaccinations was studied – the immunogen contained a V3 loop fragment and a tetanus toxin helper epitope [Zafiropoulos (1997)]

---

396 10F10 gp160(304–320) gp120(MN) RKRIHIGPGRAFYTT L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* gp120

Ab type: V3 **References:** [Duarte (1994)]

- 2C4: Putative epitope lies within IHIGPGRAFYT – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte (1994)]

---

397 2C4 gp160(304–320) gp120(MN) RKRIHIGPGRAFYTT L (MN) Vaccine murine(IgG2a)

Vaccine: *Vector/type:* peptide *Strain:* MN

Ab type: V3 **References:** [Duarte (1994)]

- 2C4: Putative epitope lies within IHIGPGRAFYT – neutralizes MN, not IIIB and SF2 – generated by multi-epitope polypeptide immunization – recognize MN and SC (TRSIHIGPGRAFYTT) peptides, lower affinity for SF2 [Duarte (1994)]

---

398 412-D (412–10D, 412, 412D) gp160(304–320) gp120(MN) RKRIHIGPGRAFYTT L HIV-1 infection human(IgG1κ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1993), Spear (1993), VanCott (1994), Fontenot (1995), Gorny (1998), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 412-D: Neutralizes MN, does not bind SF2 or HXB2 – not reactive with hexa or heptapeptides by Pepscan [Gorny (1993)]
- 412-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 412-D: Called 412–10D – relatively rapid dissociation and weak homologous neutralization [VanCott (1994)]
- 412-D: Called 412 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)]
- 412-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 412-D has a relatively fast dissociation, thus low affinity among V3 MAbs [Gorny (1998)]
- 412-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 412-D was bound only to B clade virions and to D clade MAL [Nyambi (1998)]
- 412-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 412-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]

- 412-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 412-D showed limited reactivity [Nyambi (2000)]

---

399 polyclonal gp160(304–320) gp120(MN) RKRIHIGPGRAFYTT L (MN ALA-1) HIV-1 infection human( )

Ab type: V3 **References:** [Spear (1994)]

- 40% of antibody in serum that can bind to native viral proteins on MN-infected cells can be blocked by the peptide RKRIHIGP-GRAFYTT, which can also block 75–95% of the complement activation on HIV infected cells [Spear (1994)]

---

400 CGP 47 439 gp160(304–322) gp120( ) L Vaccine human(Ig)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Liou (1989), Safrit (1993), Gunthard (1994), Gauduin (1998), Jacobson(1998)]

- CGP 47 439: passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus – CGP 47 439 is a BAT123-human Ig chimera [Safrit (1993)]
- CGP 47 439: Phase I/IIA clinical trial studying multidose tolerability, immunogenicity and pharmacokinetic responses – GP 47 439 was well tolerated, serum t_1/2 was 8–16 days, and a virus burden reduction was noted in some patients [Gunthard (1994)]
- CGP 47 439: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – in this circumstance complement activation provided a protective advantage [Gauduin (1998)]
- CGP 47 439: Review of passive immunotherapy, summarizing [Gunthard (1994)] in relation to other studies [Jacobson(1998)]

---

401 178.1 (178.1.1) gp160(305–309) gp120(305–309 BH10) KSiRI L Vaccine murine(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* yeast derived gp160

Ab type: V3 **Donor:** C. Thiriart, Smith Kline and MRC AIDS reagent project
References: [Thiriart (1989), Back (1993), Moore & Ho(1993), Cook (1994)]

- 178.1: Reacts to gp120 and gp160 in RIPA EIA and immunoblot [Thiriart (1989)]
- 178.1: Called 178.1.1 – conformational, does not bind well to denatured gp120 [Moore & Ho(1993)]
- 178.1: gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662–675 is ELDKWANLWNWFNI [Back (1993)]
- 178.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook (1994)]
- 178.1: UK Medical Research Council AIDS reagent: ARP331

402	257-D (257, 257–2-D-IV, 257-D-IV, 257, 257–2D, 257D)	gp160(dis 305–309)	gp120(dis MN)	KRIHI		L	HIV-1 infection	human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Cavacini (1993a), Spear (1993), D'Souza (1994), VanCott (1994), Stamatatos & Cheng-Mayer(1995), D'Souza (1995), Zolla-Pazner (1995), Schutten (1995a), Schutten (1995b), Fontenot (1995), Wisnewski (1996), Schutten (1996), Schutten (1997), Stamatatos (1997), Hill (1997), LaCasse (1998), Yang (1998), Gorny (1998), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Nyambi (2000), Park (2000), York (2001)]

- 257-D: Called 257–2-D-IV – potent neutralizing MAb [D'Souza (1991)]
- 257-D: Reacts with MN, NY5, CDC4 and SF2, does not cross-react with RF, WM52, or HXB2 [Karwowska (1992b)]
- 257-D: Neutralizes MN – binds SF2: KSIYI – specificity: MN, SF2, NY5, RF. [Gorny (1993)]
- 257-D: Additive MN or SF2 neutralization when combined with CD4 binding site MAb F105 – does not neutralize RF [Cavacini (1993a)]
- 257-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG – complement mediated virolysis of MN, but not in the presence of sCD4 [Spear (1993)]
- 257-D: Included a multi-lab study for antibody characterization and assay comparison – best NAb against MN, but not IIIB [D'Souza (1994)]
- 257-D: Potent MN neutralization, slow dissociation constant [VanCott (1994)]
- 257-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 better revealed this V3 epitope on TCLA SF2 and dual tropic (MU3) viruses than on macrophage tropic isolates [Stamatatos & Cheng-Mayer(1995)]
- 257-D: Called 257-D-IV – could neutralize MN and closely related JRCSF, but not 2 B subtype and 1 D subtype primary isolates in a multi-laboratory study involving 11 labs [D'Souza (1995)]
- 257-D: In serotyping study using flow-cytometry, bound only to virus with KRIHI [Zolla-Pazner (1995)]
- 257-D: Only inhibition of SI phenotype virus, and strong enhancement of NSI phenotype chimeric viruses, that incorporated different envs from the same donor [Schutten (1995a)]
- 257-D: Comparable affinity for SI and NSI viruses, in contrast to MAb MN215 [Schutten (1995b)]
- 257-D: 257-D is V H5 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]
- 257-D: IIIB neutralizing MAbs *in vitro* fail to neutralize in a mouse model *in vivo* [Schutten (1996)]
- 257-D: Neutralized (>90%) an SI-env chimeric virus and enhanced (>200%) an NSI-env chimeric virus [Schutten (1997)]
- 257-D: Binds less extensively than MAb 391–95D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes less potently than 391–95D – stronger neutralization of primary macrophage targets than PBMC [Stamatatos (1997)]

- 257-D: Called 257 – gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]
- 257-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 257-D: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang (1998)]
- 257-D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 257-D has a slow dissociation, thus the highest affinity among V3 MAbs [Gorny (1998)]
- 257-D: Called 257D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391–95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 257-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 257-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 257-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 257-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)]
- 257-D: Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium Streptococcus gordonii which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized S. gordonii expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG2a in mice [Oggioni (1999)]
- 257-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 257-D showed intermediate reactivity [Nyambi (2000)]
- 257-D: Called 257D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 257-D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization, suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]
- 257-D: UK Medical Research Council AIDS reagent: ARP3023
- 257-D: NIH AIDS Research and Reference Reagent Program: 1510

403	311–11-D (311–11D, 311, 311D, 311-D)	gp160(305–313)	gp120( )	KRIHIGP	L	HIV-1 infection	human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1991), Gorny (1993), Spear (1993), Gorny (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 311–11-D: Neutralizes MN – binds SF2: KSIYIGP [Gorny (1993)]
- 311–11-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 311–11-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 311–11-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 311–11-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 311–11D showed weak reactivity [Nyambi (2000)]

404	41148D	gp160(305–313)	gp120(MN)	KRIHIGP	L	HIV-1 infection	human(IgG1)

Ab type: V3 **References:** [Pinter (1993b), Alsmadi & Tilley(1998)]

- 41148D: Neutralizes less potently than 4117C, reacts with MN, IIIB, SF2 [Pinter (1993b)]
- 41148D: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, comparable to 4117C, however 41148D is 10x less efficient at neutralization, showing ADCC and neutralization don‘t always correlate [Alsmadi & Tilley(1998)]

405	391/95-D (391–95D, 391.5, 391/95D)	gp160(dis 305–318)	gp120(dis MN)	KRIHIGPGRAFY	L	HIV-1 infection	human(IgG1κ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1991), Gorny (1993), Fontenot (1995), Stamatatos & Cheng-Mayer(1995), Seligman (1996), Stamatatos (1997), Stamatatos & Cheng-Mayer(1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Ly & Stamatatos(2000), Park (2000)]

- 391/95-D: Neutralizes MN – binds to SF2, not IIIB [Gorny (1993)]
- 391/95-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 better revealed this V3 epitope on macrophage tropic and dual tropic (MU3) viruses, but not in TCLA SF2 [Stamatatos & Cheng-Mayer(1995)]

- 391/95-D: Competition ELISAs with serial deletions estimated the epitope to be KRIHIGPGRAFY – unconstrained peptide had higher affinity than cyclic [Seligman (1996)]
- 391/95-D: Called 391–95D – binds more extensively than MAb 257-D on the surface of HIV-1 isolates SF162 and SF128A – neutralizes more potently than 257-D – stronger neutralization of primary macrophage targets than PBMC – binding post-gp120-sCD4 association related to anti-V3 Abs neutralizing capacity [Stamatatos (1997)]
- 391/95-D: Called 391–95D – deleting the V2 loop of neutralization-resistant HIV-1 isolate SF162 does not abrogate its replication in PBMC or macrophages, but it enhances its neutralization sensitivity to sera from patients with B clade infection up to 170-fold, and also enhances sensitivity to sera from clades A through F – deletion of V1 or V2 did not enable neutralization by V3 MAbs 391–95D or 257D [Stamatatos & Cheng-Mayer(1998)]
- 391/95-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 391/95-D: Called 391.5 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 391/95-D: Called 391–95D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447-D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]
- 391/95-D: Called 391/95D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

---

406 Aw gp160(305–320) gp120(Gun-1wt) KSITIGPGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Aw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Aw gives weak neutralization of both wildtype and v strains [McKnight (1995)]

---

407 Bw gp160(305–320) gp120(Gun-1wt) KSITIGPGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Bw: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – Bw gives weak neutralization of only the wildtype strain, does not bind to variant [McKnight (1995)]

---

408 DO142–10 (DO 142–10) gp160(305–320) gp120(MN) KRIHIGPGRAFYTT L HIV-1 infection human Fab(IgG1)

Ab type: V3 **References:** [Seligman (1996), Ditzel (1997), Parren (1997b), Parren & Burton(1997), Parren (1998a), Sullivan (1998a)]

- DO142–10: Fab fragment – competition ELISAs with serial deletions defined the epitope KRIHIGPGRAFYTT [Seligman (1996)]
- DO142–10: Phage expression libraries panned against MN peptide were used to select Fab DO142–10 – Fab binds MN gp120, but not a primary isolate rec gp120 [Ditzel (1997)]
- DO142–10: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- DO142–10: Binds to gp120 MN and an MN V3 peptide with equal affinity, but binds a consensus B peptide and JRCSF less well, and to IIIB gp120 not at all [Parren & Burton(1997)]
- DO142–10: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different that Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- DO124–10: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – Fab Ab fragment DO124–10 also enhances YU2 entry, ruling out Fc interactions or Env cross-linking as a mechanism – while DO124–10 enhances YU2 entry 6-fold, it neutralizes HXBc2 under identical conditions [Sullivan (1998a)]

---

409 Dv gp160(305–320) gp120(Gun-1v) KSITIGSGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Dv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)]

---

410 Fv gp160(305–320) gp120(Gun-1v) KSITIGSGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Fv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)]

---

411 Gv gp160(305–320) gp120(Gun-1v) KSITIGSGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Gv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)]

---

412 Hv gp160(305–320) gp120(Gun-1v) KSITIGSGRAFHAI L Vaccine rat( )

Vaccine: *Vector/type:* peptide *Strain:* Gun-1 *HIV component:* V3

Ab type: V3 **References:** [McKnight (1995)]

- Hv: Rat antibodies were raised against V3 peptides that represent either the wildtype (wt), or brain-cell tropic variant (v) of the isolate Gun-1 – neutralization of only the variant strain, does not bind to wildtype [McKnight (1995)]

---

413 50.1 (R/V3–50.1, Fab 50.1) gp160(306–310) gp120(MN) RIHIG L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf, Repligen Corporation, Cambridge, MA

References: [D'Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Rini (1993), Bou-Habib (1994), VanCott (1994), Robert-Guroff (1994), Moore (1994b), VanCott (1995), Fontenot (1995), Seligman (1996), Berman (1997), LaCasse (1998), Stanfield (1999), Hoffman (1999), Park (2000), York (2001)]

- 50.1: Called R/V3–50.1 – potent neutralizing of lab strains[D'Souza (1991)]
- 50.1: Epitope defined by peptide reactivity and changes affinity with amino acid substitutions – epitope RIHIGP [White-Scharf (1993)]
- 50.1: No synergistic neutralization of MN when combined with CD4BS MAb F105 – isotype stated to be IgG2a [Potts (1993)]
- 50.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 and 50.1 Fab fragments – epitope KRIHIGP [Ghiara (1993)]
- 50.1: Crystal structure of V3 loop bound to 50.1 – light chain binds just to the left of GPG, heavy chain binds further to the left [Rini (1993)]
- 50.1: No neutralization of primary isolate JR-CSF – greater affinity for and neutralization of T cell tropic strain T-CSF, derived from JR-CSF [Bou-Habib (1994)]
- 50.1: Potent MN neutralization, slow dissociation rate [VanCott (1994)]
- 50.1: Chimeric MN V3 loop in an HXB2 background allows increased FACS signal, Ab affinity, and viral neutralization [Robert-Guroff (1994)]
- 50.1: Shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (1994b)]
- 50.1: Used to monitor HIV-1 Env expression in infected H9 cells [VanCott (1995)]
- 50.1: Competition ELISAs with serial deletions produced comparable estimate of epitope length to crystal structure and alanine substitution – KRIHIGP [Seligman (1996)]
- 50.1: Binds to 6/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]
- 50.1: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 50.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different Fabs were bound [Stanfield (1999)]
- 50.1: A CD4-independent viral variant of IIIB, IIIBx, was generated on CXCR4-expressing cells – IIIBx exhibited enhanced neutralization of by CD4i MAbs and by polyclonal human sera but not by anti-V3 MAb 50.1 [Hoffman (1999)]

- 50.1: Called R/V3–50.1 – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes – 50.1 could only neutralize the sensitive form [Park (2000)]
- 50.1: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – the dissociation constant, Kd of 50.1 for the cell associated primary and TCLA Envs was equal, 7nM [York (2001)]
- 50.1: NIH AIDS Research and Reference Reagent Program: 1289

---

414 polyclonal gp160(306–318) gp120(NY5) KKGIAIGPGRTLY (IgM)

Ab type: V3 **References:** [Metlas (1999b), Metlas (1999a)]

- Auto-Abs that react with the V3 loop of NY5 are present in the sera of HIV- individuals, and are predominantly IgM [Metlas (1999b)]

---

415 BAT123 (BAT-123, CGP 47 439) gp160(306–322) gp120(308–322 HXB2) RIRIQRGPGRAFVTIGK L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* inactivated virus *Strain:* IIIB *HIV component:* virus

Ab type: V3 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Fung (1987), Liou (1989), Fung (1990), Moore & Ho(1993), Safrit (1993), Thali (1993), Pirofski (1993), Gauduin (1995), Sattentau & Moore(1995), Poignard (1996a), Andrus (1998), Parren (1998a), Gauduin (1998)]

- BAT123: CGP 47 439 is a BAT123 chimera that has a human IgG1 Fc domain
- BAT123: Anti-idiotypic MAb, AB19–4i, stimulates anti-anti-ID which neutralizes MN and IIIB [Fung (1990)]
- BAT123: Called BAT-123 – conformational, does not bind well to denatured gp120 – not reactive with SF-2 gp120 – does not inhibit HIV-1 sera from binding to IIIB gp120 [Moore & Ho(1993)]
- BAT123: Passive transfer to Hu-PBS-SCID mice confers protection against challenge with homologous cell-free virus [Safrit (1993)]
- BAT123: Variable region sequenced – heavy chain: V 3660-SB32, D unknown, J H3 – light chain: V κ21, J κ2 [Pirofski (1993)]
- BAT123: Passive transfer of BAT123 to hu-PBL-SCID mice 1 hour prior to inoculation with HIV-1 LAI, or up to four hours post-exposure, could protect mice from infection – the protection, like the MAb, was specific for the viral strain LAI [Gauduin (1995)]
- BAT123: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strain [Sattentau & Moore(1995)]
- BAT123: Epitope described as RGPGRAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus (BAT123 less so than the others), mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- BAT123: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]

- BAT123: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- BAT123: Post-exposure passive transfer of murine BAT123 can confer protection to hu-PBL-SCID mice challenged with HIV-1 LAI – this protection is not elicited by CGP 47 439, a BAT123 chimera that has a human IgG1 Fc domain, suggesting that the protection is mediated by complement – the protective ability of BAT123 is lost when mice were treated with cobra venom factor, which inactivates serum complement – IgG1 does not fix complement efficiently so an IgG2 MAb might perform better [Gauduin (1998)]

---

416 838-D (838) gp160(307–311) Env(RF) KSITK L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1997), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Gorny (2000), Nyambi (2000)]

- 838-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 838-D was cross-reactive with V3 peptides from clade A and C, and could bind to 5/8 B clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)]
- 838-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 838-D bound B clade virions but had limited cross-reactivity with other clades, with low levels of binding to A and D virions [Nyambi (1998)]
- 838-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to many A, B, C and F peptides, poor binding to D and E [Zolla-Pazner (1999a)]
- 838-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 838-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10 fold preference for the oligomer [Gorny (2000)]
- 838-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 838-D showed intermediate reactivity [Nyambi (2000)]

---

417 1006–15D (1006) gp160(307–312) gp120(RF) KSITKG no HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1006–15D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – was somewhat cross-reactive with V3 peptides from clade A, C and other B clade V3 peptides, but not E clade [Gorny (1997)]
- 1006–15D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A peptides – no binding was observed with D and E peptides [Zolla-Pazner (1999a)]
- 1006–15D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]

- 1006–15D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1006–15D showed strong cross-reactivity [Nyambi (2000)]

---

418 782-D (782) gp160(307–312) Env(RF) KSITKG L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 782-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 782-D was not cross-reactive with V3 peptides from clade A or E, but could bind to 3/8 B clade V3 peptides, and 1/2 C clade V3 peptides – 50% neutralization of RF was obtained [Gorny (1997)]
- 782-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several B and F peptides, one C peptide, and some reactivity with A and D peptides [Zolla-Pazner (1999a)]
- 782-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 782-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 782-D showed intermediate reactivity [Nyambi (2000)]

---

419 908-D (908, 908–12D) gp160(307–312) gp120(RF) KSITKG L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 908-D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 908-D was not cross-reactive with V3 peptides from clade E, but could bind to 6/8 B clade V3 peptides, 2/4 A clade, and 1/2 C clade – 50% neutralization of RF was obtained [Gorny (1997)]
- 908-D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed strong binding to several A, B, C and F peptides, and poor binding to E and D peptides [Zolla-Pazner (1999a)]
- 908-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 908-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross -reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 908-D showed strong cross-reactivity, but achieved only 50% neutralization on 2/5 isolates tested [Nyambi (2000)]

---

420 1027–15D (1027, 1027-D, 1027D) gp160(307–313) Env(RF) KSITKGP no HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1997), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 1027–15D: Five human MAbs against were derived from HIV-infected North American subjects after selection by the V3 RF peptide – 1027–15D was not cross-reactive with V3 peptides from clade A or E, but could bind to 3/8 B clade V3 peptides, and 1/2 C clade V3 peptides [Gorny (1997)]
- 1027–15D: Review of clade specificity and anti-V3 HIV-1-Abs – this Ab showed moderate binding to several B and F peptides, one C peptide, and was not reactivity with A, D and E peptides [Zolla-Pazner (1999a)]
- 1027–15D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 838, 782, 1027, 908, and 1006, all selected with RF V3 peptide – the core amino acids KSITK tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 1027–15D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 1027–15D showed strong cross-reactivity [Nyambi (2000)]

---

421 F19.26–4 gp160(307–319) gp120(312–324 LAI) IRIQRGPGRAFVT L Vaccine murine(IgG2aκ)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Boudet (1994)]

- F19.26–4: Strain specific – used to raise anti-idiotype antibodies [Boudet (1994)]

---

422 F19.48–3 gp160(307–319) gp120(312–324 LAI) IRIQRGPGRAFVT L Vaccine murine(IgG2aκ)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Boudet (1994)]

- F19.48–3: Strain specific – used to raise anti-idiotype antibodies [Boudet (1994)]

---

423 F19.57–11 gp160(307–319) gp120(312–324 LAI) IRIQRGPGRAFVT L (LAI) Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Boudet (1991), Boudet (1994), Boudet (1995)]

- F19.57–11: MAb F19.57–11 is strain specific for LAI – used to raise anti-idiotype rabbit antibodies (called 57-B Ab2) [Boudet (1994)]
- F19.57–11: Anti-anti-idiotypic antibodies (Ab3) were raised in BALBc mice that had greater breadth of reactivity than the original F19.57–11 (Ab3 could also recognize 1282 and SF2, with aa TRK(R or S)IYIGPGRA(WY or FH)T) [Boudet (1995)]

---

424 M77 gp160(307–320) gp120(IIIB) IRIQRGPGRAFVTI L HIV-1 infection human(IgG)

Ab type: V3 **Donor:** Advanced BioScience Laboratories, Rockville, MD, commercial

References: [Pal (1992), di Marzo Veronese (1992), di Marzo Veronese (1993), Watkins (1993), Cook (1994), DeVico (1995), Denisova (1995), Watkins (1996), Denisova (2000)]

- M77: IIIB-specific MAb, immunoprecipitates deglycosylated form [di Marzo Veronese (1992)]
- M77: Antibody binding to viral isolates from IIIB infected lab worker followed through time – A to T substitution resulted in the loss of neutralization and native gp120 binding, but not peptide binding [di Marzo Veronese (1993)]
- M77: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]

- M77: Reacted with both reduced and non-reduced covalently cross-linked gp120-CD4 complex [DeVico (1995)]
- M77: Conformational rearrangements upon binding of M77 to gp120 generates novel epitopes called metatopes [Denisova (1995)]
- M77: Stated to be a murine MAb – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – M77 neutralization was only slightly reduced by this mutation [Watkins (1993)]
- M77: Used M77 bound to gp120 as an immunogen – analysis of polyclonal and monoclonal (62 MAbs were generated) response suggests the M77-gp120 immunogen generated MAbs to more linear epitopes than gp120 alone or gp120 bound to CD4 [Denisova (1996)]
- M77: Native M77 is highly strain specific, and V3 binding is primarily dependent on its heavy chain – a light chain switched Fab version of M77 could recognize HIV-1 strains that had substitutions on the left side of the V3 loop – R in GPGR is likely to be critical for binding [Watkins (1996)]
- M77: M77 is highly strain specific for IIIB, but anti-idiotypic Abs directed against M77 can in turn elicit an Ab response with expanded HIV cross-reactivity – this mechanism may serve to prolong the primary response and to counter-balance viral immune evasion by mutation [Denisova (2000)]

---

425 SP.BAL114 gp160(308–317) gp120(BAL) SIHIGPGRAF L murine?(IgG2aκ)
Ab type: V3 **References:** [Arendrup (1995)]
- Authors suggest that during *in vivo* immunoselection of escape virus, the V3 domain gains increasing resemblance to that of lab strains [Arendrup (1995)]

---

426 SP.SF2:104 gp160(308–317) gp120(SF2) SIYIGPGRAF L HIV-1 infection (IgG2aκ)
Ab type: V3 **References:** [Arendrup (1993), Arendrup (1995)]
- SP.SF2:104: Anti-V3 antibody that could neutralize primary virus isolated from a time point of neutralization resistance of autologous virus [Arendrup (1993)]
- SP.SF2:104: Authors suggest that during *in vivo* immunoselection of escape virus, the V3 domain gains increasing resemblance to lab strains [Arendrup (1995)]

---

427 polyclonal gp160(308–319) gp120(304–318 LAI) RIHIGPGRAFYT HIV-1 infection human(IgG,IgM)
Ab type: V3 **References:** [Langedijk (1995)]
- Polylconal sera from six individuals tested for reactivity against a panel of peptides based on autologous sequences provide evidence for immunological escape mutations in the tip of the V3 loop [Langedijk (1995)]

---

428 19b gp160(308–320) gp120( ) -I—-G–FY-T L HIV-1 infection human(IgG1)
Ab type: V3 **Donor:** James Robinson, University of Connecticut, Storrs
References: [Scott (1990), Moore (1994b), Moore (1994a), Sattentau(1995), Moore (1995b), Moore (1995a), Moore & Ho(1995), Gauduin (1996), Wu (1996), Trkola (1996a), D'Souza (1997), Binley (1997a), Fouts (1997), Ugolini (1997), Boots (1997), Parren (1997b), Mondor (1998), Parren (1998a), Trkola (1998), Binley (1999), Park (2000), Kolchinsky (2001)]
- 19b: V3 loop binding MAb that is more broadly clade cross-reactive than most (binds to 19/29 clade B and 10/12 clade E gp120s) [Moore (1994b)]

- 19b: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore (1994a)]
- 19b: Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)]
- 19b: Binds to some gp120s from clades A,B,C,E, and F – weakly neutralized some B and one C clade virus [Moore (1995b)]
- 19b: Despite broad gp120 binding reactivity, not broadly neutralizing [Moore (1995a)]
- 19b: Review: more broadly cross-reactive than anti-V3 tip MAb 447-D [Moore & Ho(1995)]
- 19b: Not as effective as IgG1b12 at neutralization *ex vivo* of virus direct from plasma of HIV-1 infected individuals [Gauduin (1996)]
- 19b: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of 19b blocks this inhibition [Wu (1996)]
- 19b: Inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 19b: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – there were four sequences with variations in the defined epitope among the 9 isolates tested [D'Souza (1997)]
- 19b: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 19b bound monomer, did not bind oligomer or neutralize JRFL [Fouts (1997)]
- 19b: Viral binding inhibition by 19b was weakly correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 19b: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 19b has an epitope involving the tip of the V3 loop, with 5 or 6 essential amino acids distributed within a 12 amino acid stretch – the previously determined binding site was confirmed -I—-G–FY-T and some tolerated variants described, the I can be I, V, or L, the Y can be Y, F, or W – probably a β-turn is required for FY or FF binding, but WY in can bind with out the context of the turn [Boots (1997)]
- 19b: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- 19b: Used as a control in this Hx10 binding and neutralizing MAb study because 19b does not bind to Hx10 [Mondor (1998)]
- 19b: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 19b: No detectable neutralizing activity among primary isolates with different co-receptor usage – some neutralization of TCLA strains [Trkola (1998)]
- 19b: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]
- 19b: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form but 19b was an exception and required around 950 ng/ml to neutralize either form [Park (2000)]

- 19b: Mutations in two glcosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to 19b [Kolchinsky (2001)]

---

429 4G10 gp160(308–322) gp120(308–322 LAI) RIQRGPGRAFVTGK Vaccine murine( )

Vaccine: *Vector/type:* HBcAg fusion *HIV component:* V3

Ab type: V3 **Donor:** Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [von Brunn (1993)]

- 4G10: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)]
- 4G10: NIH AIDS Research and Reference Reagent Program: 2534

---

430 5F7 gp160(308–322) gp120(308–322 LAI) RIQRGPGRAFVTGK Vaccine murine( )

Vaccine: *Vector/type:* HBcAg fusion *HIV component:* V3

Ab type: V3 **Donor:** Dr. Albrecht von Brunn, Max-von-Pettenkofer-Institut, Ludwig-Maximilians-Universitat Munchen, Germany
References: [von Brunn (1993)]

- 5F7: A 25 amino acid V3-loop sequence fused to HBcAg enhanced V3 immunogenicity [von Brunn (1993)]
- 5F7: NIH AIDS Research and Reference Reagent Program: 2533

---

431 G3-523 gp160(308–322) gp120(308–322) RIQRGPGRAFVTIGK murine( )
Ab type: V3 **References:** [Matsushita (1988), Jagodzinski (1996)]

- G3-523: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits G3-523 binding [Jagodzinski (1996)]

---

432 MN215 gp160(308–322) gp120(MN) RIHIGPGRAFYTTKN L HIV-1 infection human(IgG1)
Ab type: V3 **References:** [Schutten (1995b)]

- MN215: Minimum epitope for MAB using the Dutch consensus is AFYTTGE, different than defined for MN – generated by EBV transformation of PBMC – displayed higher affinity for NSI than for SI glycoproteins – amino acids HIGP were essential for binding [Schutten (1995b)]

---

433 Nea 9301 gp160(308–323) gp120(IIIB) RIQRGPGRAFVTIGKI murine( )
Ab type: V3 **Donor:** Dupont, commercial
References: [Wagner (1996)]

---

434 4117C gp160(309–315) gp120( ) IXIGPGR L HIV-1 infection human(IgG1λ)
Ab type: V3 **References:** [Tilley (1991a), Tilley (1992), di Marzo Veronese (1993), Pinter (1993a), Pinter (1993b), Alsmadi & Tilley(1998)]

- 4117C: Potent neutralizing activity against MN, SF-2, and NY-5 – synergy with CD4BS MAb 1125H [Tilley (1991a)]

- 4117C: Neutralizes SF2 and MN synergistically combined with anti-CD4 binding site discontinuous MAb [Pinter (1993a), Tilley (1992)]
- 4117C: Binds V3 loop – does not immunoprecipitate soluble gp120, does react with gp120 on intact virions [Pinter (1993b)]
- 4117C: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against MN and SF2, but not IIIB and RF [Alsmadi & Tilley(1998)]

---

435 419-D (419, 419D) gp160(309–315) gp120(MN) IHIGPGR L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Karwowska (1992b), Gorny (1993), Spear (1993), Fontenot (1995), Nyambi (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 419-D: MN, NY5 and SF2 strain specific, does not cross-react with RF, CDC4, WM52 or HXB2 [Karwowska (1992b)]
- 419-D: Neutralizes MN – binds SF2: IYIGPGR [Gorny (1993)]
- 419-D: Mediated deposition of complement component C3 on HIV infected cells, enhanced by second Ab binding, rabbit anti-human IgG [Spear (1993)]
- 419-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 419-D bound to 3/4 B clade virions, and to D clade MAL [Nyambi (1998)]
- 419-D: Review of clade specificity and anti-V3 HIV-1-Abs – epitope is described as KRIHIGP [Zolla-Pazner (1999a)]
- 419-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 419-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 419-D showed intermediate reactivity, and no neutralization when tested against five strains – discrepancy between the epitope as described in earlier papers and as described here, KRIHIGP [Nyambi (2000)]

---

436 453-D (453) gp160(309–315) gp120(MN) IHIGPGR L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1991), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 453-D: Neutralizes MN – binds SF2: IYIGPGR – specificity: MN, SF2, NY5, RF [Gorny (1993)]
- 453-D: Moderate homologous neutralization, moderately slow dissociation rate [VanCott (1994)]
- 453-D : Called 453, epitope described as KRIHIGPGR – the tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)]
- 453-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 453-D : MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – MAb 268, with a previously defined core epitope identical to 453 (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]

- 453-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 453-D showed intermediate reactivity [Nyambi (2000)]

---

437 504-D (504, 504–10D) gp160(309–315) gp120(MN) IHIGPGR L HIV-1 infection human(IgG1κ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1993), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 504-D – Neutralizes MN – binds SF2: IYIGPGR [Gorny (1993)]
- 504-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 504-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 504-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 504-D showed weak reactivity [Nyambi (2000)]

---

438 83.1 (MAb 83.1) gp160(309–315) gp120(SF2) IYIGPGR L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf, Repligen Corporation, Cambridge, MA
References: [White-Scharf (1993), Potts (1993), Jelonek (1999), Keller & Arora(1999), Binley (1999)]

- 83.1: Neutralizes SF2 [White-Scharf (1993)]
- 83.1: Study of synergism of neutralization and binding comparing F105 and sCD4 with the V3 MAbs: 50.1, 59.1, 83.1, and 58.2 – synergy was observed, and the data suggest that binding of one ligand (F105) can increase the binding of the second (*e.g.* V3 loop MAbs) due to conformational changes [Potts (1993)]
- 83.1: Maternally transferred anti-V3 loop MAb selectively inhibits the anti-V3 loop Ab component of the IgG response to rgp120 SF2 in 21 day old BALBc mice [Jelonek (1999)]
- 83.1: 19 day old mice injected with 83.1 have a shift in IgG1 response away from the V3 loop upon vaccination, without decreasing the total IgG anti-gp120 response, suggesting that prior treatment with a MAb can mask immunogenic sites and shift the immune response to vaccination [Keller & Arora(1999)]

- 83.1: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]

---

439 5023B | gp160(309–316) | gp120(309–316 BH10) | IQRGPGra | no | Vaccine | murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991)]

- 5023B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

---

440 F58/D1 (F58) | gp160(309–316) | gp120(IIIB) | IxxGPGRA | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990), Broliden (1991), Moore (1993b), Millar (1998), Jackson (1999)]

- F58/D1: Binding to native gp120 1–3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]
- F58/D1: The interaction of a 17-amino-acid neutralizing microantibody (MicroAB) based on F58 and HIV-1 env was studied by electrospray ionization mass spectrometry [Millar (1998)]
- F58/D1: A 17 amino acid MicroAB was made from the third complementarity-determining region of the heavy chain of MAb – F58 neutralized 5x's more efficiently in terms of mass than the original MAb, 32-fold less on a molar basis – neutralization does not involve initial attachment, but fusion and events in early infection [Jackson (1999)]

---

441 P1/D12 | gp160(309–316) | gp120( ) | IxxGPGRA | L | Vaccine | murine(IgG)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990), Moore (1993b)]

- P1/D12: Binding to native gp120 1–3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]

---

442 P4/D10 (P4D10) | gp160(309–316) | gp120( ) | IxxGPGRA | L | Vaccine | murine(IgG1κ)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990), Broliden (1990), Broliden (1991), Marks (1992), Moore (1993b), Arendrup (1993), Hinkula (1994), Jacobson(1998), Schonning (1998), Schonning (1999)]

- P4/D10: Neutralizing and ADCC activity [Broliden (1990)]
- P4/D10: Variable domain sequenced and is identical to F58/H3 [Marks (1992)]
- P4/D10: Binding to native gp120 3 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]
- P4/D10: Primary isolates from different time points from one individual were not susceptible to neutralization by P4/D10 [Arendrup (1993)]
- P4/D10: Used for passive immunotherapy in four late-stage HIV-infected patients – the serum level of p24 did not decrease in any of these four – see also MAb F58/H3 [Hinkula (1994)]
- P4/D10: Review of passive immunotherapy, summarizing [Hinkula (1994)] in relation to other studies [Jacobson(1998)]
- P4/D10: Called P4D10 – In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU – Ab binding site was suggested to be 314–323 of BRU [Schonning (1998)]
- P4/D10: Called P4D10 – the stoichiometry of MAb neutralization was tested and the data indicated that binding for neutralization was was incremental not all or none, *i.e*., each envelope oligomer binds a single MAb and each Env oligomer bound reduces the chances of infection – MAb BC1071 was used for virion quantitation – P4D10 binds only to Env with a glycosylation site mutation at the base of the V3 loop A308T [Schonning (1999)]

---

443 IIIB-13 V3 (1044–13, IIIB-V3–13) gp160(309–317) gp120(308–316 IIIB) IQRGPGRAF L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB

Ab type: V3 **References:** [Laman (1992), Laman (1993), D'Souza (1994), Watkins (1993)]

- IIIB-13 V3: Also known as 1044–13 and as IIIB-V3–13 (J. P. Moore, per. comm.)
- IIIB-13 V3: Neutralizes IIIB but not MN [Laman (1992)]
- IIIB-13 V3: Included in a panel of antibodies used in a multi-lab study for antibody characterization and assay comparison, some neutralization of strains other than IIIB [D'Souza (1994)]
- IIIB-13 V3: Called IIIB-V3–13 – a neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – IIIB-V3–13 neutralization was only slightly reduced by this mutation [Watkins (1993)]
- IIIB-13 V3: UK Medical Research Council AIDS reagent: ARP3046
- IIIB-13 V3: NIH AIDS Research and Reference Reagent Program: 1727

---

444 IIIB-34 V3 (IIIB-V3–34) gp160(309–317) gp120(308–316 IIIB) IQRGPGRAF L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB

Ab type: V3 **References:** [Laman (1992), Laman (1993)]

- IIIB-34 V3: Neutralizes IIIB but not MN – QXGPG are critical amino acids for binding by Pepscan analysis [Laman (1992)]

- IIIB-34 V3: Called IIIB-V3–34 – IIIB strain specific neutralization – binding is reduced somewhat by DTT or SDS-DTT, enhanced by NP40, but binds to native and denatured gp120 [Laman (1993)]
- IIIB-34 V3: UK Medical Research Council AIDS reagent: ARP3047

---

445 A47/B1 gp160(309–318) gp120(307–316 IIIB) IQRGPGRAFV L Vaccine murine(IgG)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990)]

---

446 D59/A2 gp160(309–318) gp120(307–316 IIIB) IQRGPGRAFV L Vaccine murine(IgG)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990)]

---

447 G44/H7 gp160(309–318) gp120(307–316 IIIB) IQRGPGRAFV L Vaccine murine(IgG)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* gp120

Ab type: V3 **References:** [Akerblom (1990)]

---

448 μ5.5 (5.5, μ5.5, Rμ5.5) gp160(309–319) gp120(MN) IHIGPGRAFYT P L murine(IgG1κ)

Ab type: V3 **References:** [Maeda (1992), Okamoto (1998)]

- μ5.5: sCD4 causes loss of IIIB type-specificity for MAb 0.5β, allowing binding and neutralization of MN, in contrast to MAb μ5.5 [Maeda (1992)]
- μ5.5: Rμ5.5 is a humanized antibody of mouse MAb m5.5 – neutralized primary isolates with similar V3 loops – passive transfer of MAb to SCID-hu or hu-PBL-SCID mice conferred protection [Okamoto (1998)]

---

449 loop 2 (Loop 2, IgG1 Loop 2) gp160(309–320) gp120( ) SISGPGRAFYTG L HIV-1 infection human Fab( )

Ab type: V3 **Donor:** D. Burton, Scripps Research Institute, La Jolla, CA
References: [Barbas III (1993), Moore (1994b), Wu (1996), Ditzel (1997), Ugolini (1997), Parren (1997b), Parren & Burton(1997), Mondor (1998), Parren (1998a), Sullivan (1998a)]

- loop2: Also known as Loop 2, IgG1 Loop 2 was a obtained by engineering Fab loop2 into an IgG1 molecule
- loop 2: Sequences of the heavy and light chain Fab variable regions were generated [Barbas III (1993)]
- loop 2: Called Loop 2 – shows modest cross-reactivity among B clade gp120s, little outside B clade [Moore (1994b)]
- loop 2: MIP-1α binding to CCR-5 expressing cells can be inhibited by gp120-sCD4 – binding of loop 2 blocks this inhibition [Wu (1996)]
- loop 2: Binds to gp120 from MN and SF2 but not LAI [Ditzel (1997)]
- loop 2: Viral binding inhibition by loop 2 MAb or Fab was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]

- loop 2: Epitope is suggested to be GPGRAF – binds to 10/17 US clade B monomeric gp120s – IgG1 form can neutralize MN and 2 primary isolates tested [Parren & Burton(1997)]
- loop 2: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- loop 2: The rank order of Fab binding affinity to monomeric gp120 (Loop 2 > 3B3 > b12 = DO8i > b11 > b3 > b14 > b13 > DO142–10 > DA48 > L17) was markedly different than Fab binding affinity to the mature oligomeric form (3B3 > b12 > DO142–10 > Loop 2 > b11 > L17 > b6 > DO8i > b14 > DA48 > b3 > b13) and binding to oligomeric form and neutralization were correlated for both Fabs and MAbs – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope – binding affinity of divalent IgG1 loop 2 is only 2-fold greater than monovalent Fab loop 2, suggesting the IgG1 form may bind with only one arm [Parren (1998a)]
- loop 2: The HIV-1 virus YU2 entry can be enhanced by MAbs binding to the CD4BS, V3 loop, and CD4i epitopes – the activation for this enhanced entry state could be conferred on HxB2 by introducing the YU2 V3 loop, or the YU2 V3 and V1/V2 loops – a similar effect is observed by sub-neutralizing concentrations of sCD4 and the effect is dependent of CCR5 – loop 2 enhances YU2 at concentrations up to 20 μg/ml [Sullivan (1998a)]

---

450 268-D (268–11-D-IV, 268D, 268, 268–11D, 268–10D, MAb 268, 268–10-D) | gp160(dis 310–315) | gp120(dis MN) | HIGPGR | L | HIV-1 infection | human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Gorny (1991), D'Souza (1991), Karwowska (1992b), Gorny (1993), Spear (1993), VanCott (1994), Stamatatos & Cheng-Mayer(1995), Zolla-Pazner (1995), Fontenot (1995), McKeating (1996), Wisnewski (1996), Stamatatos (1997), LaCasse (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Beddows (1999), Oggioni (1999), Laisney & Strosberg(1999), Hioe (2000), Nyambi (2000), Park (2000), York (2001)]

- 268-D: Called 268–11-D-IV – strain specific weakly neutralizing [D'Souza (1991)]
- 268-D: Reacts with MN, NY5, CDC4, RF and SF2, does not cross-react with WM52 or HXB2 [Karwowska (1992b)]
- 268-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny (1993)]
- 268-D: Mediated deposition of complement component C3 on HIV infected cells, but not in the presence of sCD4 [Spear (1993)]
- 268-D: Moderate dissociation rate and homologous neutralization titer [VanCott (1994)]
- 268-D: Serotyping study using flow-cytometry, if H of HIGPGR was substituted in virus, 268-D did not bind [Zolla-Pazner (1995)]
- 268-D: The binding of conformation-dependent anti-V2, anti-V3, and anti-CD4BS MAbs to monomeric and virion-associated gp120 from HIV-1 isolates with differences in cell tropism was studied – V3 loop epitopes were less accessible to Ab binding on the virion surface than in the gp120 monomer, particularly for macrophage-tropic isolates SF162 and SF128a, relative to T-cell tropic SF2 – sCD4 association with gp120 did not influence the binding of 268-D to virion-associated gp120, although sCD4 binding did alter epitope exposure for other anti-V3 MAbs [Stamatatos & Cheng-Mayer(1995)]
- 268-D: Failed to neutralize HXB2 and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]

- 268-D: 268-D is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]
- 268-D: Poor reactivity against HIV-1 isolates SF162 and SF128A and no neutralization, in contrast to MAbs 391/95-D and 257-D [Stamatatos (1997)]
- 268-D: A T-cell line-adapted (TCLA) derivative of SI primary isolate 168P acquired the ability to be neutralized by anti-V3 MAbs – the primary isolate could use either CCR5 or CXCR4, and was not neutralized when infection was directed via either pathway, however the TCLA derivative uses CXCR4 only and is neutralized [LaCasse (1998)]
- 268-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 268-D: Peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group – MAb 453, with an identical core epitope to 268 based on prior experiments (HIGPGR), was not part of this reactivity group, illustrating that context can be critical [Zolla-Pazner (1999b)]
- 268-D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – 268-D bound rgp120 W61D but could only neutralize the W61D isolate following T-cell line adaptation [Beddows (1999)]
- 268-D: Called 268–11D – Study of a live-vector mucosal vaccine that expresses HIV-1 V3 domains using the bacterium Streptococcus gordonii which can express heterologous Ag and can colonize the oral cavity and vagina of mice – 268-D and 257-D recognized S. gordonii expressing the V3 domain of MN – the vaccine stimulated V3-specific IgG2a in mice [Oggioni (1999)]
- 268-D: Called MAb 268 – To identify potential mimotopes of V3, a hexapeptide phage library was screened with MAb 268 – two hexamers were identified, HLGPGR or KAIHRI that bind to 268 with the same binding site as the V3 loop and inhibit 268 MN gp120 – KLH conjugated hexamer KAIHRI stimulates Abs in rabbits that cross-react with ML gp120 [Laisney & Strosberg(1999)]
- 268-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52-D and 268–10-D did not effect proliferation [Hioe (2000)]
- 268-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 268-D showed weak reactivity [Nyambi (2000)]
- 268-D: Called 268D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 268-D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – one of the TCLA V3 viruses 320SI-C3.3 shows reduced binding with this MAb, the sequence of the epitope in 320SI is HIGPGR and in 320SI-C3.3 is RIGPGR [York (2001)]
- 268-D: UK Medical Research Council AIDS reagent: ARP3024
- 268-D: NIH AIDS Research and Reference Reagent Program: 1511

451	386-D (386, 386–10D, 386D)	gp160(310–315)	gp120(MN)	HIGPGR	L	HIV-1 infection	human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Karwowska (1992b), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 386-D: Neutralizes MN – binds SF2: YIGPGR – specificity: MN, SF2, NY5, RF, CDC4 [Gorny (1993)]
- 386-D: Slow dissociation rate, potent homologous neutralization [VanCott (1994)]
- 386-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 386-D: Peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 386-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 386-D showed intermediate reactivity [Nyambi (2000)]

452	5042A	gp160(310–315)	gp120(310–315 BH10)	QrGPGR	L	Vaccine	murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991), Gorny (1991)]

- 5042A: Generation and fine mapping of murine MAbs [Langedijk (1991)]

453	5042B	gp160(310–315)	gp120(310–315 BH10)	QRGPGr	no	Vaccine	murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991)]

- 5042B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

454	418-D (418, 418D)	gp160(310–316)	gp120(MN)	HIGPGRA	L	HIV-1 infection	human(IgG1κ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Karwowska (1992b), Gorny (1993), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 418-D: MN strain specific, does not cross-react with SF2, NY5, RF, CDC4 WM52 or HXB2 [Karwowska (1992b)]
- 418-D: Neutralizes MN, does not bind to SF2 or HXB2 [Gorny (1993)]
- 418-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 418-D: Called 418 – MAb peptide-reactivity pattern clustered with immunological related MAbs: 391.5, 412 and 418, all selected with MN V3 peptide – the core amino acids HIGPGR tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]

- 418-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 418-D showed intermediate reactivity [Nyambi (2000)]

---

455 5021 gp160(310–316) gp120( ) QrGPGRa L Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Durda (1988), Durda (1990), Langedijk (1991), Moore (1993b)]

- 5021: Generation and fine mapping of murine MAbs [Langedijk (1991)]
- 5021: Binding to native gp120 100–300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]

---

456 5025B gp160(310–316) gp120(310–316 BH10) QRGPGra no Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991)]

- 5025B: Generation and fine mapping of murine MAbs [Langedijk (1991)]

---

457 5042 gp160(310–316) gp120( ) QRGPGRA L Vaccine murine( )

Vaccine: *Vector/type:* peptide

Ab type: V3 **References:** [Durda (1988), Durda (1990), Moore (1993b)]

- 5042: Binding to native gp120 100–300 fold greater than to denatured – 314G/W substitution abolishes binding, changes outside the loop have little effect [Moore (1993b)]

---

458 110.3 gp160(310–317) gp120(308–328 BRU) QRGPGRAF L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: V3 **References:** [Thomas (1988), Evans (1989), Langedijk (1992), Pirofski (1993), Connelly (1994)]

- 110.3: Included as a control [Evans (1989)]
- 110.3: MAb variable region sequenced – heavy chain: V 7138(40), D deletion, J H4 – light chain: V κ21(47), J κ2 [Pirofski (1993)]
- 110.3: An anti-idiotypic MAb generated against 110.3 both mimics and binds to V3, suggesting that the V3 loop may associated with itself [Connelly (1994)]

---

459 110.4 gp160(310–317) gp120(308–328 BRU) QRGPGRAF L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: V3 **Donor:** Genetic Systems Corp, Seattle WA, E. Kinney-Thomas
References: [Thomas (1988), Thali (1992b), Langedijk (1992), Thali (1993), Pirofski (1993), Arendrup (1993), Thali (1994), Boudet (1994), Connelly (1994), McDougal (1996), Valenzuela (1998), Cao (1997), Guillerm (1998)]

- 110.4: 313 P/S substitution in the V3 region disrupts binding [Thali (1992b)]
- 110.4: MAb variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J H2 – light chain: V κ21, J κ2 [Pirofski (1993)]
- 110.4: Primary isolates from different time points from one individual were not susceptible to neutralization by 110.4 [Arendrup (1993)]
- 110.4: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 110.4: An anti-idiotypic MAb generated against 110.3 also blocks binding of 110.4 [Connelly (1994)]
- 110.4: Neutralizes HIV-1 LAI [McDougal (1996)]
- 110.4: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11–20 is through inhibition of viral binding to the cell [Valenzuela (1998)]
- 110.4: Virus with the V1-V2 loop deleted was viable and more susceptible to neutralization by CD4i MAb 17b, and anti-V3 MAbs 1121, 9284, and 110.4, but not to and CD4BS MAb F105 or sCD4 [Cao (1997)]
- 110.4: Used for flow cytometry in a study of the anti-CD4, CDR3 loop MAb called 13B8.2, in a study of HIV-1 induced programmed cell death [Guillerm (1998)]

---

460 110.5 gp160(310–317) gp120(308–328 BRU) QRGPGRAF L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: V3 **Donor:** E. Kinney-Thomas or Genetic Systems, Seattle WA

References: [Thomas (1988), Moore (1990), Cordell (1991), Sattentau & Moore(1991), Langedijk (1992), McKeating (1992a), Pirofski (1993), Moore (1993b), Thali (1993), Klasse (1993a), Sattentau (1995), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), McDougal (1996), Jeffs (1996), Binley (1997a), Ugolini (1997), Parren (1998a)]

- 110.5: Did not induce dissociation of gp120, as sCD4 did – discrepancy with [Poignard (1996a)], that was suggested to be due to MAb interference with detection, as the gp120-MAb complex was denatured in the Poignard study [Moore (1990)]
- 110.5: Binding insensitive to gp120 reduction [Cordell (1991)]
- 110.5: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 110.5: Variable region sequenced – heavy chain: V 3660-SB32, D closest to DSP2.3, 2.4 and .6, J H2 – light chain: V κ21, J κ2 [Pirofski (1993)]
- 110.5: Thrombin cleavage of V3 loop between R-315 and A-316 abrogates binding – can inhibit C4 region antibody which has conformational requirements (G3-299) – binding to native gp120 100–300 fold greater than to denatured [Moore (1993b)]
- 110.5: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 110.5 is not affected [Reitz (1988), Klasse (1993a)]
- 110.5: Pretreatment of HX10-infected H9 cells with sCD4 decreases signal from 110.5 at 37 degrees due to dissociation of gp120-gp41 [Sattentau (1995)]
- 110.5: Binds with high affinity to monomer and oligomer, rapid association and potent neutralization of lab strains – neutralizes cell-free Hx10 [Sattentau & Moore(1995)]

- 110.5: Reciprocal binding inhibition with other anti-V3 MAbs – enhances binding of some anti-V2 MAbs – binding enhanced by some CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.5: V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- 110.5: Neutralizes HIV-1 LAI [McDougal (1996)]
- 110.5: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 110.5: Viral binding inhibition by 110.5 was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 110.5: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

---

461 58.2 gp160(310–317) gp120(MN) HIGPGRAF L Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Repligen Corp.
References: [White-Scharf (1993), Potts (1993), Moore (1994b), Seligman (1996), Stanfield (1999), York (2001)]

- 58.2: Epitope defined by peptide reactivity and changes in affinity with amino acid substitutions – 4/7 primarily isolates were neutralized [White-Scharf (1993)]
- 58.2: Did not synergistically neutralize MN in combination with MAb F105 – there was synergistic neutralization when combined with sCD4 [Potts (1993)]
- 58.2: Modest cross-reactivity among B clade gp120s, little outside B clade – core epitope as I-IHIG [Moore (1994b)]
- 58.2: Competition ELISAs with serial deletions produced longer estimates of epitope length, RIHIGPGRAFY, than Alanine substitution, suggesting significance of non-contact residues [Seligman (1996)]
- 58.2: The crystal structure of Fab 58.2 bound to V3 loop peptides was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound – 58.2's epitope was defined as KRKRIHIGPGRAFY [Stanfield (1999)]
- 58.2: 58.2's epitope was noted to be IGPGRAF – Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]

---

462 537-D (537) gp160(311–315) gp120(MN) IGPGR L HIV-1 infection human(IgG1λ)

Ab type: V3 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Karwowska (1992b), Gorny (1992), Gorny (1993), VanCott (1994), Fontenot (1995), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Nyambi (2000)]

- 537-D: Reacts with MN, NY5, CDC4, RF, WM52 and SF2, but does not cross-react with HXB2 [Karwowska (1992b)]
- 537-D: MN type specific neutralization observed – binds SF2, also IGPGR [Gorny (1992), Gorny (1993)]
- 537-D: Moderate homologous neutralization, relatively rapid dissociation constant [VanCott (1994)]
- 537-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]

- 537-D: MAb peptide-reactivity pattern clustered with immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 537-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 537-D showed weak reactivity [Nyambi (2000)]

---

463 5020 | gp160(311–316) | gp120(311–316 BH10) | RGPGRA | no | Vaccine | murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **References:** [Langedijk (1991)]

- 5020: Generation and fine mapping of murine MAbs [Langedijk (1991)]

---

464 5023A (5023, NEA-9205, NEA 9205) | gp160(311–317) | gp120(311–317 BH10) | RgPGRAF | L | Vaccine | murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **Donor:** Paul Durda, Du Pont de Nemours and Co
References: [Langedijk (1991), D'Souza (1991), Back (1993), Rovinski (1995), Schonning (1998)]

- 5023A: Generation and fine mapping of murine MAbs [Langedijk (1991)]
- 5023A: Called 5023 – Langedijk also has an MAb called 5023B – strong cross-reactive neutralizing MAb [D'Souza (1991)]
- 5023A: Called 5023 – Langedijk also has an MAb called 5023B – gp41 amino acid substitutions 668 (N/S) and 675 (I/M) in gp41 interfere with 5023s neutralization potency, region 662–675 is ELDKWANLWNWFNI [Back (1993)]
- 5023A: Called 5023 in this paper – Used to precipitate gp160 in immunoblots in a study examining the feasibility of using unprocessed gp160 glycoprotein as an immunogen [Rovinski (1995)]
- 5023A: Called NEA-9205 – The N306 glycan of the V3 loop makes the tip of the V3 loop inaccessible to this MAb in oligomeric Env, loss of this glycan enhances neutralization sensitivity [Schonning (1998)]

---

465 110.6 | gp160(311–318) | gp120(BRU) | RGPGRAFV | L (weak) | Vaccine | murine(IgG1λ)

Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: V3 **References:** [Thomas (1988), Pirofski (1993), Langedijk (1992)]

- 110.6: Variable region sequenced – heavy chain: V J558–146b.1α, D closest to DSP16.2, J H3 – light chain: V λ1, J λ1 [Pirofski (1993)]

---

466 polyclonal | gp160(311–318) | gp120(MN) | IGPGRAFY | L | Vaccine | murine(IgG2a)

Vaccine: *Vector/type:* B. abortus complex *Strain:* SF2, MN *HIV component:* gp120

Ab type: V3 **References:** [Golding (1995)]

- Ab is evoked even in mice depleted of CD4+ cells

467	10/36e	gp160(311–321)	gp120(311–321 HXB10)	RGPGRAFVTIG	L (HXB10)	Vaccine	rat(IgG2a)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993b), Peet (1998)]

- 10/36e: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]
- 10/36e: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 10/36e binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

468	10/54 (10/54ow/6i/6i)	gp160(311–321)	gp120(311–321 HXB10)	RGPGRAFVTIG	L (HXB10)	Vaccine	rat(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993a), McKeating (1993b), Peet (1998)]

- 10/54: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]
- 10/54: Studied in the context of a neutralization escape mutant [McKeating (1993a)]
- 10/54: Called 10/54ow/6i/6i: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind, but anti-V3 MAb 10/54 binding was dramatically diminished by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]

469	11/85b (11/85b/14I/14I)	gp160(311–321)	gp120(311–321 HXB10)	RGPGRAFVTIG	L (HXB2)	Vaccine	rat(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: V3 **References:** [McKeating (1992a), McKeating (1993b)]

- 11/85b: Binding to virion gp120 enhanced by sCD4 [McKeating (1992a)]

470	polyclonal	gp160(311–322)	gp120(MN)	IGPGRAFYTTKN	L (MN ALA-1)	Vaccine	guinea pig( )

Vaccine: *Vector/type:* human rhinovirus 14 *Strain:* MN *HIV component:* V3

Ab type: V3 **References:** [Smith (1998)]

- The tip of the MN V3 loop (IGPGRAFYTTKN) was inserted into cold-causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies – chimeric viruses elicited potent NAbs against ALA-1 and MN [Smith (1998)]

471	0.5β (0.5 β, 0.5β)	gp160(311–324)	gp120(316–330 HXB2)	RGPGRAFVTIGKIG	L (IIIB)	Vaccine	murine(IgG1κ)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* Env

Ab type: V3 **Donor:** Shuzo Matsushita or Toshio Hattori of Kumamoto University
References: [Matsushita (1988), Skinner (1988b), Skinner (1988a), Reitz (1988), Nara (1990), D'Souza (1991), Matsushita (1992), Emini (1992), Maeda (1992), McKeating (1992a), Sperlagh (1993), di Marzo Veronese (1993), Moore (1993b), Klasse (1993a), Watkins (1993), Cook (1994), Thali (1994), Okada (1994), Boudet (1994), Broder (1994), Zvi (1995b), Zvi (1995a), Jagodzinski (1996), Warrier (1996), McDougal (1996), Jeffs (1996), Huang (1997), Zvi (1997), Wyatt (1997), Faiman & Horovitz(1997), Fortin (2000), Jagodzinski & Trzeciak(2000), Tugarinov (2000), Zvi (2000)]

- 0.5β: Type-specific neutralization of IIIB – does not neutralize MN or RF [Matsushita (1988), Skinner (1988b)]
- 0.5β: Emergence of virus resistant to MAb 0.5β and autologous sera neutralization in IIIB infected chimps [Nara (1990)]
- 0.5β: Potent neutralizing activity [D'Souza (1991)]
- 0.5β: Chimeric mouse-human MAb Cβ1 was constructed by combining the human Cgamma1 and Ckappa constant regions with the 0.5β murine MAb – ADCC and neutralizing activity[Matsushita (1992)]
- 0.5β: sCD4 causes loss of IIIB type-specificity, allowing binding and neutralization of MN, in contrast to MAb μ5.5 [Maeda (1992)]
- 0.5β: Monoclonal anti-idiotype antibodies that mimic the 0.5β epitope were generated [Sperlagh (1993)]
- 0.5β: Neutralization of virus carrying an A to T substitution (contrast with MAb M77) [di Marzo Veronese (1993)]
- 0.5β: Binding to native gp120 100–300 fold greater than to denatured [Moore (1993b)]
- 0.5β: The gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to some antiserum and conformationally sensitive neutralizing MAbs – neutralization efficiency of 0.5β is not affected [Reitz (1988), Klasse (1993a)]
- 0.5β: A neutralization escape mutant (HXB2 A281V) was selected by growth of HXB2 in the presence of broadly neutralizing sera – of the MAbs tested , 0.5β neutralization was the most profoundly affected by this mutation [Watkins (1993)]
- 0.5β: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – this MAb can inhibit gp120 binding to GalCer *in vitro* [Cook (1994)]
- 0.5β: gp41 mutation that confers resistance to neutralization by anti-CD4 binding site antibodies does not reduce neutralizing efficiency of this V3 region MAb [Thali (1994)]
- 0.5β: Binding domain aa 310–319: RGPGRAFVTIGKIG – mutations in the V3 loop from basic residues can destroy virus infectivity and syncytium formation: R306T, R309T and R313G can also reduce binding of V3 MAbs with two different binding sites: 9284 and 0.5β [Okada (1994)]
- 0.5β: Type-specific neutralization of IIIB – does not neutralize SF2 [Broder (1994)]
- 0.5β: The interactions of the peptide RKSIRIQRGPGRAFVT 0.5β were studied by NMR, and hydrophobic interactions between the two Is and the V form the base of a 12 amino acid loop with GPGR at the apex[Zvi (1995b)]
- 0.5β: NMR of 0.5β bound NNTRKSIRIQRGPGRAFVTIGKIG suggests that the bound amino acids are in the region SIRIQRGPGRAFVT [Zvi (1995a)]
- 0.5β: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits 0.5β binding – 0.5β epitope described as GPGRAFVTIG [Jagodzinski (1996)]
- 0.5β: Synergistic neutralization of HIV-1 when combined with anti-V2 MAb C108G [Warrier (1996)]
- 0.5β: Deletion of the V1V2 regions did not affect anti-V3 Abs ability to bind when compared to intact rec gp120 [Jeffs (1996)]
- 0.5β: Relative to the native peptide, an O-linked α-galactosamine modified V3 peptide enhanced binding to 0.5 β, while an N-linked beta-glucosamine modified peptide showed reduced binding [Huang (1997)]

- 0.5β: The structure of a 17 amino acid V3 peptide bound to the Fab was studied using NMR [Zvi (1997)]
- 0.5β: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- 0.5β: The Fv fragment was purified and the temperature dependence and effect of mutations was studied [Faiman & Horovitz(1997)]
- 0.5β: Host encoded intercellular adhesion molecule (ICAM-1) is incorporated by the HIV-1 virion and enhances viral infectivity – ICAM-1 does not modify virus sensitivity to antibodies 0.5β or 4.8D or sCD4, but neutralizing ability of F105 was diminished in ICAM bearing virions in the presence of lymphocyte function-association antigen-1 (LFA-1) Ab [Fortin (2000)]
- 0.5β: MAbs 0.5β and G3-42 were used to study synthesis of oligomeric and monomeric forms of Env – inhibition of glycosylation by tunicamycin results in reduction of oligomeric gp120 at the cell surface and of monomer in the cytoplasm – neither MAb recognized non-glycosylated Env precursor [Jagodzinski & Trzeciak(2000)]
- 0.5β: 14/18 residues of peptide P1053, RKSIRIQRGPGRAFVTIG, were shown to be involved in the Ab recognition site using NMR – QRGPGR forms a beta-hairpin turn at the center of the binding pocket [Tugarinov (2000)]
- 0.5β: NMR and mutation cycles were employed to generate a model of the peptide-antibody complex, showing aa residues that interact or do not contribute to the binding of MAb 0.5β Fv with the peptide – F96(L) of 0.5β binds to Pro13, H52(H) interacts with Ile7, Ile9, Gln10, and D56(H) interacts with Arg11 of the V3 loop peptide – RGPG retains hairpin conformation binds in the center of a groove [Zvi (2000)]
- 0.5β: UK Medical Research Council AIDS reagent: ARP3025
- 0.5β: NIH AIDS Research and Reference Reagent Program: 1591

---

472 Cβ1 gp160(311–324) gp120(316–330 HXB2) RGPGRAFVTIGKIG L Vaccine human(IgG1)

Vaccine: *Vector/type:* protein *Strain:* IIIB *HIV component:* Env

Ab type: V3 **References:** [Emini (1992)]

- Cβ1: passive transfer to chimpanzees confers protection against challenge with homologous cell-free virus – mouse 0.5β human IgG1 chimera [Emini (1992)]

---

473 NM-01 gp160(312–315) gp120(MN) GPGR L Vaccine murine(IgG)

Vaccine: *Vector/type:* human rhinovirus 14 *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** M. Terada
References: [Ohno (1991), Yoshida (1997), Smith (1998)]

- NM-01: Resistance mutation selected by propagation of molecular cloned isolate in the presence of NM-01 [Yoshida (1997)]
- NM-01: The tip of the MN V3 loop was inserted into cold-causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and NM-01 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith (1998)]

---

474 1026 gp160(312–317) gp120(MN) GPGRAF L Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* MN *HIV component:* gp120

Ab type: V3 **References:** [Nakamura (1993), Bou-Habib (1994)]

- 1026: Bound diverse strains, neutralizing activity against MN, close to GPGRAF [Nakamura (1993)]
- 1026: Greater affinity for T cell-tropic strain T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF [Bou-Habib (1994)]

---

475 1034 gp160(312–317) gp120(MN) GPGRAF L Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* MN *HIV component:* gp120

Ab type: V3 **References:** [Bou-Habib (1994), Berman (1997)]

- 1034: Greater affinity for T cell tropic T-CSF, derived from JR-CSF, than to the primary isolate JR-CSF, close to GPGRAF [Bou-Habib (1994)]
- 1034: Binds to 5/7 isolates from breakthrough cases from a MN gp120 vaccine trial [Berman (1997)]

---

476 59.1 (R/V3–59.1) gp160(312–317) gp120(308–313 MN) GPGRAF L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* MN *HIV component:* V3

Ab type: V3 **Donor:** Mary White-Scharf and A. Profy, Repligen Corporation
References: [D'Souza (1991), White-Scharf (1993), Potts (1993), Ghiara (1993), Bou-Habib (1994), D'Souza (1994), Seligman (1996), Ghiara (1997), Smith (1998), Stanfield (1999), York (2001)]

- 59.1: Called R/V3–59.1 – potent neutralizing MAb [D'Souza (1991)]
- 59.1: Epitope defined by peptide reactivity and binding affinity with amino acid substitutions – GPGRAF [White-Scharf (1993)]
- 59.1: Synergistic neutralization of MN when combined with sCD4 or the CD4BS MAb F105 [Potts (1993)]
- 59.1: Crystal structure of a 24 amino acid peptide from the V3 loop bound to 59.1 Fab fragment – contact residues IGPGRAF [Ghiara (1993)]
- 59.1: Greater affinity for T-cell tropic strain T-CSF than the primary isolate JR-CSF, from which T-CSF was derived [Bou-Habib (1994)]
- 59.1: Multi-lab study for antibody characterization and assay comparison – neutralizes MN and IIIB [D'Souza (1994)]
- 59.1: Competition ELISAs with serial deletions produced longer estimate of epitope length than x-ray crystallography or Alanine substitution, RIHIGPGRAFYTT, suggesting significance of non-contact residues [Seligman (1996)]
- 59.1: A conformationally restricted analog of the tip of the V3 loop was constructed and bound with Fab 59.1 – crystal structure shows interactions between 59.1 and an MN peptide and 59.1 and the modified peptide are similar, but NMR studies reveal that the modified peptide is more ordered in solution, retaining the Fab bound form [Ghiara (1997)]
- 59.1: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 59.1 was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith (1998)]
- 59.1: The crystal structure of V3 loop peptides bound to Fabs was obtained – conformational changes in the tip of the V3 loop (GPGR) were observed when different MAbs were bound [Stanfield (1999)]
- 59.1: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]

---

477 polyclonal gp160(312–317) gp120(316–321) GPGRAF Vaccine rabbit(Ig)

Vaccine: *Vector/type:* polyepitope, protein *HIV component:* gp160 *Stimulatory Agents:* BSA

Ab type: V3 **References:** [Lu (2000b), Lu (2000a)]

- High titer response to ELDKWA and RILAVERYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA, a weak response to GPGRAFY – immunization with CG-(ELDKWA-GPGRAFY)_2-K was also tried, yielding a strong Ab response to ELDKWA, weak to GPGRAF – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]

---

478 10E3 gp160(312–318) gp120(317–323 IIIB) GPGRAFY Vaccine mouse(IgG)

Vaccine: *Vector/type:* peptide in keyhole limpet hemocyanin *Strain:* IIIB *HIV component:* V3

Ab type: V3 **References:** [Tian (2001)]

- 10E3: Peptides GPGRAFY and ELDKWAG were conjugated to keyhole limpet hemocyanin and used to raise mouse monoclonal Ab – MAb hybridomas were generated with defined specificity – 10E3 binds to the peptide GPGRAFY and to rgp160 [Tian (2001)]

---

479 polyclonal gp160(312–318) gp120(317–323) GPGRAFY Vaccine murine, rabbit( )

Vaccine: *Vector/type:* peptide *HIV component:* V3 *Stimulatory Agents:* BSA

Ab type: V3 **References:** [Yu (2000)]

- High levels of epitope-specific Abs were induced by the peptide-BSA conjugates C-(GPGRAF)_4-BSA or C-(TRPNNNTRKSIRIQRGPGRAFYTIG KI)-BSA but not by rgp160 vaccine [Yu (2000)]

---

480 N11–20 (110-H) gp160(312–320) gp120(317–325) GPGRAFVTI L (LAI) murine(IgG1κ)

Ab type: V3 **Donor:** J. C. Mazie, Hybridolab, Institut Pasteur
References: [Valenzuela (1998)]

- N11–20: Neutralization of LAI in CEM cells by anti-V3 MAbs 110.4 and N11–20 is through inhibition of virus binding to the cell [Valenzuela (1998)]

---

481 5025A (5025) gp160(313–317) gp120(313–317 BH10) pgRAF L Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *Strain:* BH10 *HIV component:* V3

Ab type: V3 **Donor:** Paul Durda, Du Pont de Nemours and Co
References: [Langedijk (1991), D'Souza (1991)]

- 5025A: Generation and fine mapping of murine MAbs [Langedijk (1991)]
- 5025: Called 5025 – strain specific weakly neutralizing [D'Souza (1991)]

---

482 N70–1.9b gp160(313–318) gp120(316–322) PGRAFY L HIV-1 infection human(IgG1)

Ab type: V3 **References:** [Robinson (1990a), Scott (1990)]

- N70–1.9b: Type specificity [Robinson (1990a)]
- N70–1.9b: Type specific neutralization, ADCC directed against MN infected cells [Scott (1990)]

483	902	gp160(313–324)	gp120(IIIB)	PGRAFVTIGKIG	L	Vaccine	murine(IgG1κ)

Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* gp160

Ab type: V3 **Donor:** Bruce Chesebro, Rocky Mountain National Laboratory, Montana
References: [Chesebro & Wehrly(1988), Laman (1993), Broder (1994), Earl (1994)]

- 902: Strain specific neutralization of HIV [Chesebro & Wehrly(1988)]
- 902: Epitope may be partially masked or altered in the oligomeric molecule [Broder (1994)]
- 902: Used as a control in a study of the influence of oligomeric structure of Env in determining the repertoire of the Ab response [Earl (1994)]
- 902: V3-BH10 peptide with loop-structure inhibits IL-2 induced T-cell proliferation, thought to be due to altering intracellular signaling, and MAb 908 can block the peptide inhibition [Sakaida1997]
- 902: NIH AIDS Research and Reference Reagent Program: 522

484	694/98-D (694/98, 694.8, 694/98D)	gp160(dis 314–317)	gp120(dis IIIB)	GRAF	L	HIV-1 infection	human(IgG1λ)

Ab type: V3 **Donor:** Drs. S. Zolla-Pazner and M. Gorny, NYU Med Center NY, NY
References: [Gorny (1991), Gorny (1992), Gorny (1993), Cavacini (1993a), Spear (1993), Gorny (1994), Laal (1994), VanCott (1994), Cook (1994), VanCott (1995), Zolla-Pazner (1995), Forthal (1995), Li (1997), Zolla-Pazner (1997), Smith (1998), Li (1998), Andrus (1998), Nyambi (1998), Schonning (1998), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Altmeyer (1999), Nyambi (2000), Park (2000)]

- 694/98-D: MAb first described [Skinner (1988b)]
- 694/98-D: Type-specific lab isolate neutralization was observed – binds with 1–3 fold greater affinity to gp120 than to peptides [Gorny (1992)]
- 694/98-D: Neutralizes MN and IIIB (GRAF) – binds SF2 (GRAF) – binding reactivity: MN, IIIB, SF2, NY5, RF, CDC4, WM52 [Gorny (1993)]
- 694/98-D: Called 694-D – complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)]
- 694/98-D: 50% neutralization of HIV-IIIB at a concentration of 0.15 μg/ml [Gorny (1994)]
- 694/98-D: Potent neutralization of IIIB – no neutralization synergy in combination with CD4 binding domain MAbs [Laal (1994)]
- 694/98-D: GRVY did not alter peptide binding – GRVI and GQAW enhanced dissociation – GQVF and GQAL did not bind [VanCott (1994)]
- 694/98-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – V3 MAbs can inhibit gp120 binding to GalCer *in vitro* – binding of GalCer to gp120 inhibited but did not completely block MAb binding[Cook (1994)]
- 694/98-D: Human HIV-1 infected sera and MAb 694/98 have high reactivity to MN and RF infected H9 cells, but Genentech rec gp120 IIIB vaccine recipients do not [VanCott (1995)]
- 694/98-D: Serotyping study using flow-cytometry – bound GRAX bearing virus in 10/11 cases – somewhat conformation dependent [Zolla-Pazner (1995)]

- 694/98-D: ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 694/98-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – could only achieve 50% neutralization alone – all Ab combinations tested showed synergistic neutralization – 694/98-D has synergistic response with MAbs F105, 15e, b12, 2F5, 17b, 2G12, and 48d, and with HIVIG [Li (1997)]
- 694/98-D: Used to study pre- and post-exposure prophylaxis Hu-PBL-SCID mice infected by an intraperitoneal injection of HIV-1 LAI – MAb half-life in plasma in mice is 9 days – 2 hours post-694/98-D mice were challenged with LAI, and at an Ab concentration of 1.32 mg/Kg, 50% of the mice were infected – one of the infected mice carried the resistant form GRTF rather than GRAF (critical amino acids for binding are GRA) – post-exposure prophylaxis was effective if delivered 15 min post-exposure, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]
- 694/98-D: The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 694/98-D was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith (1998)]
- 694/98-D: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 694/98-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 694/98-D bound only to B and D clade virions and had limited cross reactivity [Nyambi (1998)]
- 694/98-D: In a study of the influence of the glycan at position 306 of the V3 loop on MAb recognition, anti-V3 MAbs were found to neutralize an HIV-BRU mutant virus that lacks the V3 loop glycan more efficiently than HIV-BRU [Schonning (1998)]
- 694/98-D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 694/98-D: MAb peptide reactivity pattern clustered with immunological related MAbs: 1108, 386, 268, 311, 257, 694.8 – the amino acids HI tended to be critical for reactivity in this group [Zolla-Pazner (1999b)]
- 694/98-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not linear V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]
- 694/98-D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 694/98-D showed intermediate reactivity [Nyambi (2000)]
- 694/98-D: Called 694/98D – six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]

---

485 9205 (NEA-9205) gp160(315–317) gp120(IIIB) RAF (core reactivity) L Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *HIV component:* V3

Ab type: V3 **Donor:** NEN, Boston MA, commercial

References: [Durda (1990), Trujillo (1993), Allaway (1993), VanCott (1994), Fontenot (1995), Schonning (1999)]

- 9205: Called NEA-9205, epitope RIQRGPGRAFVTIGK – reacts with three human brain proteins of 35, 55, 110 kd molecular weight – similar to 9284 – RAF is the core reactivity [Trujillo (1993)]
- 9205: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]
- 9205: Neutralizes IIIB but not MN – significantly slower dissociation constant for IIIB than MN [VanCott (1994)]
- 9205: Called NEA-9205 – the stoichiometry of MAb neutralization was tested and the data indicated that binding for neutralization was was incremental not all or none, *i.e.*, each envelope oligomer binds a single MAb and each Env oligomer bound reduces the chances of infection – 9205 binds only to Env with a glycosylation site mutation in the V3 loop, A308T [Schonning (1999)]

---

486 110.I gp160(316–322) gp120(316–322) AFVTIGK L Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *HIV component:* gp120

Ab type: V3 **Donor:** F. Traincard, Pasteur Institute, France
References: [Moore (1993b), Moore (1994c), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Wyatt (1997), Parren (1998a)]

- 110.I: Binds to carboxy-terminal side of the V3 loop – inhibits binding of C4 region MAb G3-299 [Moore (1993b)]
- 110.I: Binds equally well to monomer and oligomer, rapid association and potent neutralization of lab strains [Sattentau & Moore(1995)]
- 110.I: Reciprocal binding inhibition with other anti-V3 and anti-C4 MAbs – and enhances binding of some anti-V2 MAbs – binding enhanced by some anti-CD4 binding site MAbs [Moore & Sodroski(1996)]
- 110.I: Epitope suggested to be RAFVTIGK – V3 MAbs 9284, BAT123, 110.5, and 110.I could each significantly increase gp120 dissociation from virus, mimicking sCD4, and expose the gp41 epitope for MAb 50–69, in contrast to anti-V2 MAbs [Poignard (1996a)]
- 110.I: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- 110.I: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

---

487 anti-HIV-2 polyclonal gp160(dis 315–318 + 329–331) gp120(dis 315–318 SBL6669 HIV-2) FHSQ...WCR Vaccine guinea pig(IgG)

Vaccine: *Vector/type:* peptide *Strain:* HIV-2 SBL6669-ISY *HIV component:* V3

Ab type: HIV-2 V3 **References:** [Morner (1999)]

- Neutralizing Abs against HIV-2 V3 are produced when peptides spanning two non-contiguous parts of the V3 loop are used for vaccination including amino acids 315–318 near the tip (FHSQ) and 329–331 (WCR) at the C-term Cys [Morner (1999)]

---

488 IIIB-V3–01 gp160(320–328) gp120(IIIB) IGKIGNMRQ no Vaccine murine(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *HIV component:* V3

Ab type: V3 **Donor:** Jon Laman
References: [Laman (1993)]

- IIIB-V3–01: Specific for carboxy-terminal flank of the IIIB V3 loop – epitope is hidden native gp120, exposed on denaturation [Laman (1993)]
- IIIB-V3–01: UK Medical Research Council AIDS reagent: ARP3046
- IIIB-V3–01: NIH AIDS Research and Reference Reagent Program: 1726

---

489 D/6D1 gp160(346–377) gp120(351–382 LAI) ASKLREQFGNNKTIIFKQSSG-GDPEIVTHSFN no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp120

Ab type: V4 **References:** [Bristow (1994)]

- D/6D1: V4 MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp120 and rgp160 [Bristow (1994)]

---

490 4D7/4 gp160(360–380) gp120(361–380 LAI) IFKQSSGGDPEIVTHSFNCGG Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: V4 **Donor:** S. Ranjbar, NIBSC, UK
References: [Moore (1994c)]

- 4D7/4: C3 region – the relative affinity for denatured/native gp120 is >10 [Moore (1994c)]
- 4D7/4: UK Medical Research Council AIDS reagent: ARP3051

---

491 36.1(ARP 329) gp160(361–381) gp120(362–381 LAI) FKQSSGGDPEIVTHSFNCGGE Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: V4 **References:** [Thiriart (1989), Moore (1994c)]

- 36.1: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P impair binding [Moore (1994c)]
- 36.1: UK Medical Research Council AIDS reagent: ARP329

---

492 C12 gp160(361–381) gp120(362–381 LAI) FKQSSGGDPEIVTHSFNCGGE Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: V4 **Donor:** George Lewis
References: [Moore & Ho(1993), Moore (1994c), Abacioglu (1994), Moore (1994d)]

- C12: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- C12: The relative affinity for denatured/native gp120 is >30 – mutations 380 G/F, 381 E/P, and 384 Y/E impair binding – also binds GEFFYCNSTQLFNS, gp120(380–393 LAI) [Moore (1994c)]
- C12: C3 region – epitope boundaries mapped by peptide scanning, core FNCGG [Abacioglu (1994)]

---

493 110.D gp160(380–393) gp120(380–393 LAI) GEFFYCNSTQLFNS no Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C3 **Donor:** F. Traincard, Pasteur Institute, France

References: [Moore (1994c), Valenzuela (1998)]
- 110.D: The relative affinity for denatured/native gp120 is >50 [Moore (1994c)]

---

494 B32 gp160(380–393) gp120(380–393 LAI) GEFFYCNSTQLFNS Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: C3 **References:** [Moore (1994c), Abacioglu (1994)]
- B32: The relative affinity for denatured/native gp120 is >100 – mutations 380 G/F, 381 G/P, 382 F/L, 384 Y/E, and 386 N/R impair binding [Moore (1994c)]
- B32: C3 region – epitope boundaries mapped by peptide scanning – FFY(core) [Abacioglu (1994)]

---

495 B15 gp160(395–400) gp120(395–400 BH10) WFNSTW Vaccine murine(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: V4 **Donor:** George Lewis
References: [Moore & Ho(1993), Moore (1993b), Abacioglu (1994)]
- B15: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- B15: Binds native BH10 gp120 with 5 fold less affinity than denatured – does not bind native or denatured MN gp120 [Moore (1993b)]
- B15: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

496 B34 gp160(395–400) gp120(395–400 BH10) WFNSTW Vaccine murine(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: V4 **References:** [Abacioglu (1994)]
- B34: V4 region – epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

497 polyclonal (VEI4) gp160(396–418) Env( ) FNSTWFNSTWSTEGSNNTEGS-DT HIV-1 infection human( )

Ab type: V4 **References:** [Carlos (1999)]
- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

---

498 7F11 gp160(397–439) gp120(IIIB) Vaccine murine( )

Vaccine: *Vector/type:* protein *HIV component:* gp120

References: [Lasky (1987), Nilsen (1996)]
- 7F11: There is another MAb with this name that binds to integrase [Nilsen (1996)]

---

499 5C2E5 gp160(422–431) gp120(406–415 IIIB) QFINMWQEVK Vaccine murine( )

Vaccine: *Vector/type:* protein *HIV component:* gp120

Ab type: C4 **Donor:** T. Gregory and R. Ward, Genentech, San Francisco
References: [Lasky (1987), Cordell (1991)]

- 5C2E5: Blocks the gp120-CD4 interaction [Lasky (1987)]
- 5C2E5: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)]

---

500 G3-211 gp160(423–437) gp120(423–437 IIIB) IINMWQKVGKAMYAP L Vaccine murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **References:** [Sun (1989)]

- G3-211, 42, 299, 508, 519, 536, 537: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun (1989)]

---

501 G3-537 gp160(423–437) gp120(423–437 IIIB) IINMWQKVGKAMYAP L Vaccine murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **References:** [Sun (1989), Ho (1991b), McKeating (1992b)]

- G3-537, 211, 299, 508, 519, 536, 42: Cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – different neutralization efficiencies [Sun (1989)]
- G3-537: Weakly neutralizing – binds to a linear binding domain of gp120, NMWQEVGKAMYAPPISG [McKeating (1992b)]

---

502 polyclonal gp160(425–436) gp120( ) NMWQEVGKAMYA L Vaccine murine(IgA)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *Stimulatory Agents:* cholera toxin adjuvant

Ab type: CD4BS **References:** [Bukawa (1995)]

- Polyclonal secretory IgA antibody raised by mucosal immunization is able to neutralize IIIB, SF2, and MN – HIV-1 neutralization may be due to the V3, CD4 or HPG30 component of the multicomponent peptide immunogen [Bukawa (1995)]

---

503 1795 gp160(425–441) gp120(425–441 IIIB) NMWQEVGKAMYAPPISG L Vaccine ( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: CD4BS **References:** [McKeating (1992b)]

- 1795: CD4 binding site – weakly neutralizing – binding inhibited by WQEVGKAMYA, GKAM may be involved [McKeating (1992b)]

---

504 G3-299 gp160(429–438) gp120(429–438 BRU) EVGKAMYAPP L Vaccine murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *HIV component:* gp120

Ab type: C4 **Donor:** M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Sun (1989), Moore (1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Ditzel (1997), Wyatt (1997), Parren (1998a)]

- G3-299: Best neutralization of IIIB in panel of 7 MAbs that bind overlapping epitope [Sun (1989)]
- G3-299: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop cleavage or insertion abolished binding [Moore (1993b)]
- G3-299: Binds with higher affinity to monomer than to oligomer, slow association rate, although faster than other C4 MAbs tested, with more potent neutralization of lab strain [Sattentau & Moore(1995)]
- G3-299: Discontinuous V3-C4 epitope, binding enhanced by a few anti-C1, anti-CD4 binding site, and V2 MAbs – binding reciprocally inhibited by anti-V3 MAbs – G3-229 enhances the binding of some anti-V2 MAbs [Moore & Sodroski(1996)]
- G3-299: Epitope described as KQIINMWQKVGKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50–69 [Poignard (1996a)]
- G3-299: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-299: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

---

505 G3-42 (G3 42) | gp160(429–438) | gp120(429–438 BRU) | EVGKAMYAPP | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun (1989), Moore (1993b), Thali (1993), Sattentau & Moore(1995), Jagodzinski (1996), Moore & Sodroski(1996), Poignard (1996a), Trkola (1996a), Binley (1997a), Binley (1999), Jagodzinski & Trzeciak(2000)]

- G3-42: Neutralization of IIIB but not RF [Sun (1989)]
- G3-42: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – G3-42, G3-299 have lower affinity than G3-508, G3-519, and G3-536 – bound native gp120, not denatured – poor peptide binding, epitope spans V3-C4 regions – 433A/L, 435Y/H and 430V/S substitutions impaired binding, V3 loop insertion abolished binding [Moore (1993b)]
- G3-42: Inhibits binding of CD4 inducible MAb 48d [Thali (1993)]
- G3-42: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-42: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS potently inhibits G3-42 binding – G3-42 epitope described as KVGKAMYAPP [Jagodzinski (1996)]
- G3-42: Inhibits binding of many anti-V3, -CD4 binding site, and -C4 region MAbs – enhances binding of some anti-V2 region MAbs [Moore & Sodroski(1996)]
- G3-42: Epitope described as KQIINMWQKVGKAMYAPPIS – binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50–69 [Poignard (1996a)]
- G3-42: Called G3 42 – Does not inhibit gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study – described as V3-C4 discontinuous epitope [Trkola (1996a)]

- G3-42: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]
- 0.5β: MAbs 0.5β and G3-42 were used to study synthesis of oligomeric and monomeric forms of Env – inhibition of glycosylation by tunicamycin results in reduction of oligomeric gp120 at the cell surface and of monomer in the cytoplasm – neither MAb recognized non-glycosylated Env precursor [Jagodzinski & Trzeciak(2000)]

---

506 G3-508 (G3 508) | gp160(429–438) | gp120(429–438 BRU) | EVGKAMYAPP | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** M. Fung and Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Sun (1989), Thali (1993), Moore (1993b), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Trkola (1996a), Binley (1997a), Parren (1998a), Binley (1998)]

- G3-508: Neutralization of IIIB and RF [Sun (1989)]
- G3-508: Inhibits binding of CD4 inducible MAb 48d [Thali (1993)]
- G3-508: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 10 fold greater affinity than native – 433A/L, 435Y/H and 430V/S substitutions impaired binding [Moore (1993b)]
- G3-508: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-508: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]
- G3-508: Binding resulted in slight gp120 dissociation from virus and exposure of the gp41 epitope for MAb 50–69 [Poignard (1996a)]
- G3-508: Called G3 508 – inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- G3-508: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-508: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

---

507 G3-519 | gp160(429–438) | gp120(429–438 BRU) | EVGKAMYAPP | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY
References: [Sun (1989), Moore & Ho(1993), Moore (1993b), D'Souza (1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Binley (1997a), Wyatt (1997), Parren (1998a), Binley (1999)]

- G3-519: Best neutralization of RF in panel of 7 MAbs that bind overlapping epitope [Sun (1989)]
- G3-519: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-519: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 5 fold greater affinity than native – 433A/L, 435Y/H, 438P/R and 430V/S substitutions impaired binding [Moore (1993b)]
- G3-519: Included in a multi-lab study for antibody characterization, and binding and neutralization assay comparison, also binds IIIB: IINMWQKVGKAMYAPP [D'Souza (1994)]
- G3-519: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-519: Non-reciprocal enhanced binding in the presence of the C5 MAb 1C1 and the C1 MAb 135/9 – reciprocal enhanced binding with some V2 MAbs. Inhibited binding the presence of some C4, V3 and CD4 binding site MAbs [Moore & Sodroski(1996)]
- G3-519: Epitope described as KVGKAMYAPP – binding resulted in slight gp120 dissociation from virus but no significant exposure of the gp41 epitope for MAb 50–69 [Poignard (1996a)]
- G3-519: Binds both gp120 and soluble gp120+gp41 complex efficiently, suggesting its gp120 epitope is not blocked by gp41 binding [Wyatt (1997)]
- G3-519: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- G3-519: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]

---

508 G3-536 | gp160(429–438) | gp120(429–438 BRU) | EVGKAMYAPP | L | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120

Ab type: C4 **Donor:** Tanox Biosystems Inc and David Ho, ADARC, NY

References: [Sun (1989), Ho (1991b), Cordell (1991), McKeating (1992b), Moore & Ho(1993), Moore (1993b), Gorny (1994), Sattentau & Moore(1995), Moore & Sodroski(1996), Poignard (1996a), Parren (1998a)]

- G3-536: Weak neutralization of IIIB and RF – cross-react with diverse strains by immunofluorescence – blocks HIV binding to CD4+ cells – epitope:IINMWQKVGKAMYAP [Sun (1989)]
- G3-536: Cross-competition with MAbs 5C2E5, ICR38.8f and ICR38.1a [Cordell (1991)]
- G3-536: Weakly neutralizing – binds to a linear determinant in the CD4 binding domain of gp120 [McKeating (1992b)]
- G3-536: Neutralizes IIIB, is reactive with SF-2 gp120, mild inhibition of HIV-1+ sera binding to IIIB gp120 [Moore & Ho(1993)]
- G3-536: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPIS, and SF-2 and MN gp120s – bound denatured with 15 fold greater affinity than native – 433A/L, 435Y/H, 438P/R, and 430V/S substitutions impaired binding [Moore (1993b)]

- G3-536: Enhances binding of anti-V2 MAb 697-D [Gorny (1994)]
- G3-536: Binds with higher affinity to monomer than to oligomer, slow association rate [Sattentau & Moore(1995)]
- G3-536: Inhibits binding of some V3, C4 and CD4 binding site MAbs, enhances binding of V2 region MAbs [Moore & Sodroski(1996)]
- G3-536: Epitope described as KVGKAMYAPP [Poignard (1996a)]
- G3-536: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]

---

509 ICR38.1a (38.1a, 388/389) | gp160(429–438) | gp120(427–436 BRU) | EVGKAMYAPP | L | Vaccine | rat(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: C4 **References:** [Cordell (1991), McKeating (1992b), McKeating (1992a), McKeating (1992c), McKeating (1993b), McKeating (1993a), Moore (1993b), Jeffs (1996), Peet (1998), Kropelin (1998)]

- ICR38.1a: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with MAbs G3-536, 5C2E5, and ICR38.8f [McKeating (1992b), Cordell (1991)]
- ICR38.1a: Unable to exert a synergistic effect in combination with V3 directed MAbs, in contrast to MAb 39.13g, that binds to a conformational epitope involved in CD4 binding [McKeating (1992a)]
- ICR38.1a: Studied in the context of a neutralization escape mutant [McKeating (1993a)]
- ICR38.1a: Unreactive with solid-phase decapeptide, competed in solution phase assay – ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore (1993b)]
- ICR38.1a: Called 38.1a – 10 to 20 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120 [Jeffs (1996)]
- ICR38.1a: The most variable amino acids in the V3 loop were replaced with serines to make the immunodominant V3 loop less immunogenic – these changes did not affect the ability of sCD4 or MAbs to V1/V2, C1 and C4 to bind – ICR38.1a was not affected by V3 serine substitutions – mice injected with serine substituted gp120 had a reduced response relative to wildtype, and no enhanced immunogenicity of conserved regions [Peet (1998)]
- ICR38.1a: Called 388/389 – anti-C1 region MAb 87–135/9 blocks gp120 interaction with CD4+ cells – blocking activity is additive when combined with antibodies which bind in the C4 region of gp120 (F105, 388/389, and b12) [Kropelin (1998)]
- ICR38.1a: UK Medical Research Council AIDS reagent: ARP388/ARP389

---

510 ICR38.8f | gp160(429–438) | gp120(429–438 BRU) | EVGKAMYAPP | L | Vaccine | rat(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* BH10 *HIV component:* gp120

Ab type: C4 **References:** [Cordell (1991)]

- ICR38.8f: Weakly neutralizing – binds linear determinant in the CD4 binding domain – cross-competition with ICR38.1a, 5C2E5, and G3-536 [Cordell (1991)]
- ICR38.8f:ICR 38.1a and ICR 38.8f were initially reported to be independent MAbs, but are actually subclones of the same MAb [Moore (1993b)]

---

511 MO86/C3 gp160(429–443) gp120(429–443) EVGKAMYAPPISGQI *in vitro* stimulation human(IgM)
Ab type: C4 **References:** [Ohlin (1992)]
- MO86: Generated in response to IIIB Env 286–467 upon *in vitro* stimulation of uninfected-donor lymphocytes [Ohlin (1992)]

---

512 13H8 gp160(431–440) gp120(412–453) GKAMYAPPIS L Vaccine murine(IgG)
Vaccine: *Vector/type:* recombinant protein *Strain:* MN
Ab type: C4 **References:** [Nakamura (1992), Nakamura (1993), Jeffs (1996)]
- 13H8: Cross blocks 5C2 in IIIB-rsgp160 ELISA – reactive with diverse strains in rgp120 ELISA [Nakamura (1992)]
- 13H8: Bound diverse strains, neutralizing activity against MN [Nakamura (1993)]
- 13H8: Binds V3 and C4 peptides (J. P. Moore, per. comm.)
- 13H8: 3 and 4.5 fold increased binding when V1/V2 or V1/V2 and V3 were deleted from gp120, respectively [Jeffs (1996)]

---

513 G45–60 gp160(431–440) gp120(429–438 BRU) GKAMYAPPIS L Vaccine murine(IgG1)
Vaccine: *Vector/type:* virus derived protein *Strain:* IIIB *HIV component:* gp120
Ab type: C4 **References:** [Sun (1989), Moore (1993b), Gorny (1994), Moore & Sodroski(1996), Jagodzinski (1996)]
- G45–60: C4 region – binds HXB2 20mer KQIINMWQKVGKAMYAPPI, decapeptide flanking peptides also bound – bound equivalently to native and denatured gp120 – 433A/L and 435Y/H (not 430V/S) substitutions impaired binding [Moore (1993b)]
- G45–60: Enhances binding of anti-V2 MAb 697-D [Gorny (1994)]
- G45–60: Non-reciprocal enhancement of G45–60 binding by some C1 and C5 antibodies – reciprocal enhancement of some V2 region MAbs – reciprocal inhibition with many MAbs that bind to the V3, C4 and CD4 binding site regions [Moore & Sodroski(1996)]
- G45–60: The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus CRDS inhibits G45–60 binding [Jagodzinski (1996)]

---

514 polyclonal gp160(432–451) gp120(42–61 LAI) KAMYAPPISGQIRCSSNITG no Vaccine murine( )
Vaccine: *Vector/type:* vaccinia *HIV component:* Env
Ab type: C4 **References:** [Collado (2000)]
- Vaccinia p14 can elicit NAbs and p39 tends to be immunodominant, so these two proteins were fused to regions of HIV-1 Env – reduced glycosylation was noted when p14 or p39 was placed in the N-term region of the fusion protein – chimeric proteins shifted the Env Ab response from V3 to either a C1 or C4 domain, depending on the construct – all chimeric Env proteins: 14kEnv, 39kEnv, and Env39k elicited a strong Ab response to the C1 region of gp120 (LFCASDAKAYDTEVHNVWAT), and Env39k mounted a strong response to the C4 region (KAMYAPPISGQIRCSSNITG) [Collado (2000)]

---

515 1662 gp160(433–439) gp120(IIIB) AMYAPPI no Vaccine ( )
Vaccine: *Vector/type:* poliovirus *HIV component:* Env
Ab type: C4 **References:** [McKeating (1992b)]
- 1662: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

516	1663	gp160(433–439)	gp120(IIIB)	AMYAPPI	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1663: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

517	1664	gp160(433–439)	gp120(IIIB)	AMYAPPI	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1664: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

518	1697	gp160(433–439)	gp120(IIIB)	AMYAPPI	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1697: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

519	1794	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1794: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

520	1804	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1804: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

521	1807	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1807: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

522	1808	gp160(433–442)	gp120(IIIB)	AMYAPPISGQ	no	Vaccine	( )

Vaccine: *Vector/type:* poliovirus *HIV component:* Env

Ab type: C4 **References:** [McKeating (1992b)]

- 1808: Did not bind to native gp120, epitope not exposed [McKeating (1992b)]

523	polyclonal (VEI5)	gp160(454–474)	Env( )	LTRDGGNNNNESEIFRPGGGD	HIV-1 infection	human( )

Ab type: V1, V2, V3, V4, V5 **References:** [Carlos (1999)]

- Antibody response to the epitopes in a vaccine construct (VEI) containing peptides from 5 hypervariable regions of gp120 was detected in the sera of HIV-1 positive subjects, including sera from 6 non-subtype B infections – serum samples from San Francisco, Canada and Puerto Rico cohort showed presence of antibodies against all five VEI hypervariable regions, but most consistently against the V3 region peptide NNNTRKSIRIGPGRAFYTTGDIGNIRQ [Carlos (1999)]

524	polyclonal	gp160(460–467)	gp120(LAI)	NNNNGSEI	HIV-1 infection, Vaccine	human( )

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Ab type: V5 **References:** [Loomis-Price (1997)]

- HIV-1+ positive individuals were given a gp160 vaccine as immunotherapy, and this region was the most reactive new epitope as measured by a modified Pepscan technique which improved sensitivity – 4/14 showed vaccine-induced reactivity [Loomis-Price (1997)]

525	CRA1(ARP 323) (CRA-1)	gp160(461–470)	gp120(451–470 LAI)	SNNESEIFRL	no	Vaccine	murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: V5C5 **Donor:** M. Page, NIBSC, UK
References: [Moore & Ho(1993), Moore (1994d), Moore (1994c), Moore & Sodroski(1996), Trkola (1996a)]

- CRA1: Bound preferentially to denatured IIIB and SF2 gp120 [Moore & Ho(1993)]
- CRA1: Some C5 mutations abrogate binding 470 P/L or G, 475 M/S, some C2 mutations enhance binding [Moore (1994d)]
- CRA1: The relative affinity for denatured/native gp120 is 24 – C5 mutations 470 P/L or G, 475 M/S impairs binding to the native gp120 – only mutation 470 P/L impairs binding to denatured [Moore (1994c)]
- CRA1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – reciprocal binding inhibition with anti-C5 antibodies 1C1 and M91 – non-reciprocal binding enhancement some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]
- CRA1: Does not neutralize JR-FL nor block gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- CRA1: UK Medical Research Council AIDS reagent: ARP323

526	M91	gp160(461–470)	gp120(451–470 LAI)	SNNESEIFRL	no	Vaccine	rat(IgG2a)

Vaccine: *Vector/type:* protein *HIV component:* Env

Ab type: V5C5 **Donor:** Fulvia di Marzo Veronese
References: [di Marzo Veronese (1992), Moore (1994c), Moore (1994d), Moore & Sodroski(1996), Ditzel (1997), Binley (1998)]

- M91: Immunoblot reactive, RIP negative, but precipitates deglycosylated gp120 – reacts with strains IIIB, 451, MN, RF, and RUTZ [di Marzo Veronese (1992)]

- M91: The relative affinity for denatured/native gp120 is 24 – mutation in position 470 P/L impairs binding [Moore (1994c)]
- M91: 470 P/L impairs binding, but not 475 D/V, in contrast to CRA1 – some C2 mutations can enhance binding [Moore (1994d)]
- M91: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of C1 and V2 antibodies – non-reciprocal binding inhibition of CD4 binding site antibodies [Moore & Sodroski(1996)]
- M91: A panel of MAbs were shown to bind with similar or greater affinity and similar competition profiles to a deglycosylated or variable loop deleted core gp120 protein (Δ V1, V2, and V3), thus such a core protein produces a structure closely approximating full length folded monomer [Binley (1998)]

---

527 9201 gp160(471–482) gp120(475–486 LAI) GGGDMRDNWRSE no murine( )

Ab type: C5 **Donor:** Du Pont
References: [McDougal (1996)]

- 9201: Does not neutralize LAI [McDougal (1996)]

---

528 1C1 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C5 **Donor:** Repligen Inc, Cambridge, MA, commercial
References: [Moore (1994c), Moore (1994d), VanCott (1995), Moore & Sodroski(1996)]

- 1C1: The relative affinity for denatured/native gp120 is 15 [Moore (1994c)]
- 1C1: C2 and V3 regions substitutions can influence binding [Moore (1994d)]
- 1C1: Linear epitope not exposed on conformationally intact gp120 [VanCott (1995)]
- 1C1: C5 region linear epitope, binds weakly to nondenatured monomeric gp120 – M91 binding was enhanced by 1C1, but 1C1 binding was inhibited by M91 – non-reciprocal binding enhancement of some C1 and V2 antibodies – non-reciprocal binding inhibition of some CD4 binding site antibodies [Moore & Sodroski(1996)]

---

529 3F5 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C5 **Donor:** S. Nigida, NCI, USA
References: [Moore (1994c)]

- 3F5: The relative affinity for denatured/native gp120 is 100 [Moore (1994c)]

---

530 5F4/1 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine( )

Vaccine: *Vector/type:* peptide *Strain:* HIV-2 ROD

Ab type: C5 **Donor:** S. Ranjbar, NIBSC, UK
References: [Moore (1994c)]

- 5F4/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>10 fold) – mutation 485 K/V impairs binding [Moore (1994c)]

---

531 660–178 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C5 **Donor:** G. Robey, Abbott Labs
References: [Moore (1994c), Moore (1994d)]

- 660–178: The relative affinity for denatured/native gp120 is >100 [Moore (1994c)]
- 660–178: ΔV1/V2 and ΔV1/V2/V3 reduce binding – C2 and C5 mutations enhance binding [Moore (1994d)]

---

532 9301 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* Env

Ab type: C5 **Donor:** Dupont, commercial
References: [Skinner (1988b), Moore & Ho(1993), Moore (1994c), Moore (1994d), Wagner (1996)]

- 9301: Bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- 9301: The relative affinity for denatured/native gp120 is 19 [Moore (1994d)]
- 9301: Wagner *et al*. claim that Nea 9301 is anti-V3 – might they have meant MAb 9305? [Wagner (1996)]

---

533 B221 (221) gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG1κ)

Vaccine: *Vector/type:* recombinant protein *Strain:* NL43 *HIV component:* gp160

Ab type: C5 **Donor:** Rod Daniels
References: [Moore & Ho(1993), Bristow (1994), Moore (1994c)]

- B221: Called 221 – bound preferentially to denatured IIIB gp120 [Moore & Ho(1993)]
- B221: MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, Micro-GenSys [Bristow (1994)]
- B221: The relative affinity for denatured/native gp120 is 12 – mutation 477 D/V impairs binding [Moore (1994c)]
- B221: Called 221 – C2 and V3 substitutions influence binding [Moore (1994d)]
- B221: UK Medical Research Council AIDS reagent: ARP301

---

534 8C6/1 gp160(471–490) gp120(471–490 LAI) GGGDMRDNWRSELYKYKVVK Vaccine murine(IgG)

Vaccine: *Strain:* LAI

Ab type: V5C5 **Donor:** S. Ranjbar, NIBSC, UK
References: [Moore (1994c)]

- 8C6/1: V5-C5 region – preferentially binds SDS-DTT denatured gp120 (>30 fold) – mutation 485 K/V impairs binding [Moore (1994c)]
- 8C6/1: UK Medical Research Council AIDS reagent: ARP3052

---

535 H11 gp160(472–477) gp120(472–477 HXB2) GGDMRD murine( )

Ab type: C5 **References:** [Pincus & McClure(1993), Pincus (1996)]

- H11: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]

---

536 W2 gp160(472–491) gp120(472–491 LAI) GGDMRDNWRSELYKYKVVKI Vaccine murine(IgG)

Vaccine: *Strain:* LAI *HIV component:* Env

Ab type: C5 **Donor:** D. Weiner, U. Penn., USA
References: [Moore (1994c)]

- W2: The relative affinity for denatured/native gp120 is 30 – mutation 485 K/V impairs binding [Moore (1994c)]

---

537 1331A gp160(dis gp120(dis 510–516) dwVVQREKR HIV-1 infection human(IgG3λ) gp160(483–508))

Ab type: C5 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Nyambi (1998), Gorny (2000), Hochleitner (2000b), Nyambi (2000)]

- 1331A: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they don‘t bind to IIIB), and to subtype D MAL [Nyambi (1998)]
- 1331A: Core epitope dwVVQREKR maps to gp120(510–516) – binding of panel of 21 MAbs to soluble oligomeric gp140 versus gp41 or gp120 monomers was compared – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)]
- 1331A: The Ab binding site was studied with epitope excision (protein is bound in native conformation to immobilized MAb, then digested with proteolytic enzymes) and extraction (protein is digested then allowed to react with Ab), followed by mass spectroscopy – two non-contiguous aa in C5 were protected, E-507 and I-487, which are thought to be located on opposite sides of hydrophobic pocket involved in gp120/gp41 interaction [Hochleitner (2000b)]
- 1331A: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495–516), bound to 18/26 [Nyambi (2000)]

---

538 M38 gp160(485–504) gp120(490–508) KYKVVKEIPLGVAPTKAKRR no Vaccine murine( )

Vaccine: *Vector/type:* virus *Strain:* IIIB *HIV component:* virus

Ab type: C5 **References:** [Beretta (1987), Grassi (1991), Lopalco (1993), DeSantis (1994), Beretta & Dalgleish(1994)]

- M38: Binds to gp120 and to a 80 kd human protein expressed on a small fraction of mononuclear cells in the lymph nodes [Beretta (1987)]
- M38: Binds to the carboxy terminus of gp120, in a gp41 binding region, and also to denatured human HLAs (antigenic homology) [Lopalco (1993)]
- M38: Infected individuals have HLA class I-gp120 cross-reactive antibodies [DeSantis (1994)]

539	Chim 1 (C-1)	gp160(487–493)	gp120(492–498 HXB2)	KVVKEIP		humanized chimpanzee( )

References: [Pincus & McClure(1993), Pincus (1996)]

- Chim 1: Binds to gp120 but not to infected cells – when linked to ricin A, the immunotoxin did not mediate cell killing – sCD4 has no effect [Pincus & McClure(1993), Pincus (1996)]

540	polyclonal	gp160(489–511)	gp120(495–516 BRU)	KIEPLGVAPTKAKRRVVQREKR	no HIV-1 infection	human( )

References: [Hernandez (2000)]

- Chimeric peptide combining two peptides gp160(495–516 and 584–612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)]

541	110.1	gp160(491–500)	gp120(491–500 LAI)	IEPLGVAPTK	no Vaccine	murine(IgG1κ)

Vaccine: *Vector/type:* infected-cell lysate *Strain:* BRU *HIV component:* virus

Ab type: C5 **Donor:** Genetic Systems Corp, Seattle WA, E. Kinney-Thomas

References: [Gosting (1987), Linsley (1988), Thomas (1988), Pincus (1991), Moore (1994c), Cook (1994), McDougal (1996), Binley (1997a), Valenzuela (1998)]

- 110.1: There is another antibody with this ID that binds to gp120, but at aa 200–217 [Pincus (1996)]
- 110.1: Referred to as 110–1 – does not inhibit CD4-gp120 binding or neutralize HIV-1 strains [Linsley (1988)]
- 110.1: Difference in the epitope: mapped to aa 421–429 (KQIINMWQE), the T1 sequence – poor efficacy as an immunotoxin when linked to RAC [Pincus (1991)]
- 110.1: The relative affinity for denatured/native gp120 is 0.7 [Moore (1994c)]
- 110.1: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 inhibit gp120 binding to GalCer but not as potently as anti-V3 MAbs – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]
- 110.1: Does not neutralize HIV-1 LAI [McDougal (1996)]
- 110.1: Does not effect LAI viral binding or entry into CEM cells [Valenzuela (1998)]

542	42F	gp160(491–500)	gp120(491–500 HXB2)	IEPLGVAPTK	no HIV-1 infection	human(IgG1λ)

Ab type: C5 **References:** [Alsmadi (1997), Alsmadi & Tilley(1998)]

- 42F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi (1997)]
- 42F: A study of 6 anti-Env MAbs and their ability to bind or direct ADCC against target cells infected with IIIB, MN, SF-2, and RF – bound and directed lysis against strains IIIB, MN, SF-2, and RF, but not a clone of MN [Alsmadi & Tilley(1998)]

543	43F	gp160(491–500)	gp120(491–500 HXB2)	IEPLGVAPTK	no HIV-1 infection	human(IgG1λ)

Ab type: C5 **References:** [Alsmadi (1997)]

- 43F: 42F and 43F were isolated from a long term non-progressor by EBV transformation of PBMC – samples were taken 14 months apart – both MAbs stained diverse strains of infected cells and directed ADCC – were more potent for ADCC if the cell was infected with HIV-1, rather than just presenting absorbed gp120 [Alsmadi (1997)]

---

544 RV110026 | gp160(491–500) | gp120(491–500 LAI) | IEPLGVAPTK | Vaccine | human( )

Vaccine: *Vector/type:* peptide *Strain:* LAI

Ab type: C5 **Donor:** Commercial, Olympus Inc
References: [Moore (1994c), Moore (1994d)]

- RV110026: Preferentially binds SDS-DTT denatured gp120 (15 fold using R1/87 as capture reagent) [Moore (1994c)]

---

545 105–306 | gp160(492–500) | gp120(498–505 HAM112, O group) | KPFSVAPTP | Vaccine | murine(IgG1κ)

Vaccine: *Vector/type:* recombinant protein *Strain:* HAM112 (group O) *HIV component:* gp160

Ab type: C-term **References:** [Scheffel (1999)]

- 105–306: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – 105–306 bound to two overlapping peptides [Scheffel (1999)]

---

546 GV1G2 | gp160(494–499) | gp120(494–499 IIIB) | LGVAPT | Vaccine | murine( )

Vaccine: *Vector/type:* protein-Ab complex *HIV component:* gp120 complexed with MAb M77

Ab type: C5 **References:** [Denisova (1996)]

- GV1G2: When anti-V3 MAb M77 was bound to gp120 and used as an immunogen, it stimulated many MAbs to linear epitopes – MAbs GV12F6 and GV3H1 are homologous to GV1G2 and were generated in the same experiment [Denisova (1996)]

---

547 750-D | gp160(498–504) | gp120(503–509) | PTKAKRR | no HIV-1 infection | human(IgG3λ)

Ab type: C-term **References:** [Forthal (1995), Hioe (2000)]

- 750-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 750-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inihibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)]

---

548 450-D (450-D-3, 450D) | gp160(498–504) | gp120(475–486 BH10) | PTKAKRR (or RRVVQRE, or MRDNWRSELYKY depending on reference) | no HIV-1 infection | human(IgG1λ)

Ab type: C5 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY
References: [Durda (1988), Karwowska (1992a), Karwowska (1992b), Spear (1993), Laal (1994), Gorny (1994), Cook (1994), Forthal (1995), Manca (1995), Li (1997), Hioe (2000), Hioe (2001), Verrier (2001)]

- 450-D: Bound to MN, SF-2 and IIIB, but was not neutralizing [Karwowska (1992a)]
- 450-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]

- 450-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)]
- 450-D: Epitope is defined as PTKAKRR [Gorny (1994)]
- 450-D: MAbs against the glycosphingolipid GalCer block HIV infection of normally susceptible CD4 negative cells from the brain and colon – MAbs against the carboxy-terminus of gp120 do not inhibit gp120 binding to GalCer – binding of GalCer to gp120 does not inhibit MAb binding [Cook (1994)]
- 450-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 450-D: Virions complexed to gp120 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 450-D: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB env – 50% neutralization could not be achieved at a maximal concentration of 6 μg/ml [Li (1997)]
- 450-D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – C5 MAbs 450-D and 750-D did not effect proliferation [Hioe (2000)]
- 450-D: CD4BS MAbs when complexed with gp120, inhibit proliferation of gp120-specific CD4 T-cells and IFN-γ production – 450-D does not have this effect and was used as a control in this study [Hioe (2001)]
- 450-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]

---

549 670-D (670) gp160(498–504) gp120(503–509) PTKAKRR no HIV-1 infection human(IgG1λ)

Ab type: C5 **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Zolla-Pazner (1995), Forthal (1995), Hill (1997), Gorny (1997), Gorny (1998), Nyambi (1998), Altmeyer (1999), Gorny & Zolla-Pazner(2000), Nyambi (2000), Verrier (2001)]

- 670-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)]
- 670-D: Not neutralizing, positive ADCC activity, and no viral enhancing activity, numbering provided suggests epitope is RRVVQRE [Forthal (1995)]
- 670-D: gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]
- 670-D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-C5 Abs 670-D and 1331A bound to 3/4 B clade viruses (they didn't bind to IIIB), and to subtype D MAL – 670-D also reacted with subtype A[Nyambi (1998)]
- 670-D: A Semliki Forest virus (SFV) expression system carrying BX08 env was used to study the conformation of gp120 – intracytoplasmic gp120 was recognized by the anti-V3 MAbs K24 and F5.5, while gp120 at the plasma membrane was detected only by conformation dependent MAbs 2G12, 670-D and 694/98D and not V3 MAbs – expression in rat brain also showed that surface-expressed Env was recognized only by the conformation-dependent antibodies and not by anti-V3 antibodies [Altmeyer (1999)]
- 670-D: A gp120 C5 MAb used as a negative control in a study of anti-gp41 MAbs [Gorny & Zolla-Pazner(2000)]
- 670-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 670-D bound 21/26, and was the most cross-reactive C5 MAb [Nyambi (2000)]

- 670-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]

---

550 polyclonal gp160(503–509) gp120(471–477) RRVVQRE Vaccine murine(IgG)

Vaccine: *Vector/type:* peptide *HIV component:* gp120

References: [Jeyarajah (1998)]

- Mice were immunized with peptide APTKAKRRVVQREKR – epitope excision and extraction combined with mass spectrometry was used to map the fine structure of epitopes recognized by polyclonal Ab to HIV-1 Env – a major epitope was identified between positions 472 and 478 [Jeyarajah (1998)];

---

551 722-D gp160(503–509) gp120(503–509) RRVVQRE no HIV-1 infection human(IgG1κ)

Ab type: C-term **References:** [Laal (1994), Forthal (1995)]

- 722-D: Not neutralizing alone, could synergize anti-CD4 binding site antibody neutralization [Laal (1994)]
- 722-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]

---

552 polyclonal gp160(503–511) gp120(508–516) RRVVQREKR HIV-1 infection human( )

Ab type: C-term **References:** [Palker (1987), Loomis-Price (1997)]

- Most HIV-1+ individuals have an antibody response to this epitope – in this study, reactivity to RRVVQREKR was used as a positive control for HIV-1+ gp160 vaccine recipients [Loomis-Price (1997)]

---

553 1131-A gp160(505–511) gp120(510–516 LAI) VVQREKR no HIV-1 infection human(IgG3λ)

Ab type: C-term **References:** [Bandres (1998)]

- 1131-A: A very high affinity antibody used in studies that demonstrate that CXCR4 can bind to gp120 in the absence of CD4-gp120 interactions, and that this binding can be enhanced by Env deglycosylation [Bandres (1998)]

---

554 858-D gp160(505–511) gp120(510–516 LAI) VVQREKR no HIV-1 infection human(IgG)

Ab type: C-term **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)

References: [Zolla-Pazner (1995), Forthal (1995), Gorny (2000), Nyambi (2000)]

- 858-D: Group specific cross-clade binding in serotyping study using flow-cytometry [Zolla-Pazner (1995)]
- 858-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 858-D: The binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)]
- 858-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 858-D bound only 4/26, the worst of all anti-C5 MAbs tested, while MAb 1331A, which shares the same core epitope (positions 495–516), bound to 18/26 [Nyambi (2000)]

---

555 989-D gp160(505–511) gp120(LAI) VVQREKR HIV-1 infection human(IgG)
Ab type: C-term **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu) (NYU Med. Center)
References: [Zolla-Pazner (1995), Gorny (2000), Nyambi (2000)]
- 989-D: In serotyping study using flow-cytometry, showed B clade specificity, but only reacted with 7/11 B clade virus [Zolla-Pazner (1995)]
- 989-D: The binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – C5 MAbs 858-D, 989-D and 1331A bound with a 5–10 fold preference for the monomer[Gorny (2000)]
- 989-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 4 C5 MAbs, 2 bound well, 2 bound weakly – MAb 989-D bound to 6/26 [Nyambi (2000)]

---

556 1A1 gp160(525–543) gp41(526–543 BH10) AAGSTMGAASMTLTVQARQ no HIV-1 infection human(IgG1κ)
Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria
References: [Buchacher (1994)]
- 1A1: Human MAb generated using EBV transformation of PBL from HIV-1+ volunteers [Buchacher (1994)]

---

557 24G3 gp160(525–543) gp41(526–543 BH10) AAGSTMGAASMTLTVQARQ no HIV-1 infection human(IgG1κ)
Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria
References: [Buchacher (1992), Buchacher (1994)]
- 24G3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)]

---

558 25C2 (IAM 41–25C2) gp160(525–543) gp41(526–543 BH10) AAGSTMGAASMTLTVQARQ no HIV-1 infection human(IgG1κ)
Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX
References: [Buchacher (1992), Buchacher (1994), Sattentau (1995)]
- 25C2: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells – binds oligomeric and monomeric gp41, and gp160 [Buchacher (1994)]
- 25C2: Called IAM 41–25C2 – Binding domain overlaps sites that are critical for gp120-gp41 association – binding is enhanced by sCD4 – binding region defined as: gp41(21–38 BH10) [Sattentau (1995)]

---

559 5F3 gp160(525–543) gp41(526–543 BH10) AAGSTMGAASMTLTVQARQ no HIV-1 infection human(IgG1κ)
Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria
References: [Buchacher (1994)]
- 5F3: Human MAb generated by electrofusion of PBL from HIV-1+ volunteers with CB-F7 cells [Buchacher (1994)]

---

560 α(566–586) gp160(561–581) gp41(566–586 BRU) AQQHLLQLTVWGIKQLQARIL HIV-1 infection human( )
References: [Poumbourios (1992)]

---

561 PC5009 gp160(572–591) gp41(577–596 BRU) GIKQLQARILAVERYLKDQQ Vaccine murine( )

Vaccine: *Vector/type:* recombinant protein *HIV component:* gp160

References: [Poumbourios (1992)]

- PC5009: Recognized only monomeric gp41 [Poumbourios (1992)]

---

562 polyclonal α(577–596) gp160(572–591) gp41(577–596 BRU) GIKQLQARILAVERYLKDQQ HIV-1 infection human plasma( )

References: [Poumbourios (1992)]

- α(577–596): Affinity purified from HIV-1+ plasma – preferentially bind oligomer [Poumbourios (1992)]

---

563 polyclonal gp160(576–592) gp41(583–599) LQARILAVERYLKDQQL HIV-1 infection human sera( )

References: [Klasse (1993b)]

- 42 HIV-1 positive human sera were tested against wildtype peptide, and peptide with substitution 589 A to T: 11/42 reacted strongly with wildtype, weakly with A589T – 31 reacted weakly with parental, even more weakly with substituted [Klasse (1993b)]

---

564 1F11 gp160(578–612) gp41(579–613 BH10) ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA no HIV-1 infection human(IgG1κ)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria

References: [Buchacher (1992), Buchacher (1994)]

- 1F11: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]

---

565 1H5 gp160(578–612) gp41(579–613 BH10) ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA no HIV-1 infection human(IgG1κ)

References: [Buchacher (1992), Buchacher (1994)]

- 1H5: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]

---

566 3D9 gp160(578–612) gp41(579–613 BH10) ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA no HIV-1 infection human(IgG1κ)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria

References: [Buchacher (1992), Buchacher (1994)]

- 3D9: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]

---

567 4B3 gp160(578–612) gp41(579–613 BH10) ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA no HIV-1 infection human(IgG1λ)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria

References: [Buchacher (1992), Buchacher (1994), Chen (1994b)]

- 4B3: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]

---

568 4D4 gp160(578–612) gp41(579–613 BH10) ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA no HIV-1 infection human(IgG1λ)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX
References: [Buchacher (1992), Buchacher (1994), Chen (1994b), Sattentau (1995), Binley (1999)]

- 4D4: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]
- 4D4: The MAbs with the broadest neutralizing activity, IgG1b12, 2G12 and 2F5, all have high affinity for the native trimer, indicating that they were raised in an immune response to the oligomer on the virion surface rather than dissociated subunits – a disulfide linked gp120-gp41 (SOS gp140) was created to mimic the native conformation of Env and explore its potential as an immunogen – SOS gp140 is recognized by NAbs IgG1b12, 2G12, and CD4-IgG2, and also by anti-V3 MAbs 19b and 83.1 – SOSgp140 is not recognized by C4 region MAbs that neutralize only TCLA strains, G3-42 and G3-519 – nor did it bind C11, 23A, and M90, MAbs that bind to gp120 C1 and C5, where it interacts with gp41 – MAbs that bind CD4 inducible epitopes, 17b and A32 were very strongly induced by CD4 in SOS gp140 – anti-gp41 MAbs that bind in the region that interacts with gp120, 7B2, 2.2B, T4, T15G1 and 4D4, did not bind to SOSgp140, in contrast to 2F5, which binds to the only gp41 epitope that is well exposed in native gp120-gp41 complexes [Binley (1999)]

---

569 4G2 | gp160(578–612) | gp41(579–613 BH10) | ARILAVERYLKDQQLLGIWGC-SGKLICTTAVPWNA | no | HIV-1 infection | human(IgG1κ)

Donor: H. Katinger, Inst. Appl. Microbiol., Vienna, Austria
References: [Buchacher (1992), Buchacher (1994)]

- 4G2: Generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]

---

570 polyclonal | gp160(579–589) | gp41(586–596 IIIB) | RILAVERYLKD | Vaccine | mouse, rabbit( )

Vaccine: *Vector/type:* peptide *HIV component:* gp41 *Stimulatory Agents:* BSA

Ab type: C-domain **References:** [Xiao (2000b)]

- Strong epitope-specific neutralizing antibody responses were induced using the peptide C(RILAVERYLKD)_2-BSA, but not full gp160 [Xiao (2000b)]

---

571 polyclonal | gp160(579–589) | gp41(586–596) | RILAVERYLKD | Vaccine | rabbit(Ig)

Vaccine: *Vector/type:* polyepitope, protein *HIV component:* gp160 *Stimulatory Agents:* BSA

Ab type: N-term **References:** [Lu (2000b), Lu (2000a)]

- High titer response to ELDKWA and RILAVERYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA, a weak response to GPGRAFY – immunization with CG-(ELDKWA-GPGRAFY)_2-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRAFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]

---

572 polyclonal | gp160(579–599) | gp41(583–604) | RILAVERYLKDQQLLGIWGCS | no | Vaccine | rabbit sera( )

Vaccine: *Vector/type:* protein *HIV component:* desialylated gp160

References: [Benjouad (1993)]

- MAbs raised against desialylated HIV-1 gp160 cross-react with HIV-2 gp140 due to immunodominant conserved epitope in gp41 [Benjouad (1993)]

573	2A2/26	gp160(579–601)	gp41(584–606 BRU)	RILAVERYLKDQQLLGIWGCS-GK	Vaccine	murine(IgG)

Vaccine: *Vector/type:* protein *HIV component:* gp41

References: [Poumbourios (1992), Poumbourios (1995)]

- 2A2/26: Immunodominant region, binds both oligomer and monomer [Poumbourios (1992)]
- 2A2/26: Δ 550–561 (Δ LLRAIEAQQHLL), a region important for oligomer formation diminishes binding, Δ (550–561 +571–581) abrogates binding [Poumbourios (1995)]

574	50–69 (SZ-50.69)	gp160(dis 579–613)	gp41(dis 579–613 BH10)	RILAVERYLKDQQLLGIWGCS-GKLI	no HIV-1 infection	human(IgG2κ)

Ab type: cluster I **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Till (1989), Pinter (1989), Gorny (1989), Xu (1991), Robinson (1991), Sattentau & Moore(1991), Eddleston (1993), Spear (1993), Laal (1994), Chen (1995), Sattentau (1995), Manca (1995), McDougal (1996), Poignard (1996a), Binley (1996), Klasse & Sattentau(1996), Stamatatos (1997), Boots (1997), Mitchell (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000), Zwick (2001b), Verrier (2001)]

- 50–69: Combined with deglycosylated A chain of ricin is toxic to lines of HIV-infected T cells (H9) and monocytes (U937) [Till (1989)]
- 50–69: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)]
- 50–69: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)]
- 50–69: The epitope is affected by the conformation conferred by the two cysteines at amino acids 598 and 604 [Xu (1991)]
- 50–69: Enhances HIV-1 infection *in vitro* – synergizes with huMAb 120–16 *in vitro* to enhance HIV-1 infection to level approaching that found in polyclonal anti-HIV serum [Robinson (1991)]
- 50–69: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 50–69: Called SZ-50.69 – binds to an epitope within aa 579–613 [Eddleston (1993)]
- 50–69: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)]
- 50–69: Epitope described as cluster I, 601–604, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)]
- 50–69: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)]
- 50–69: Preferentially binds oligomer – binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)]
- 50–69: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 50–69: Does not neutralize HIV-1 LAI [McDougal (1996)]
- 50–69: Prebinding of anti-V3, and CD4i MAbs 48d and 17b, but not anti-V2 neutralizing MAbs, expose the 50–69 epitope [Poignard (1996a)]
- 50–69: Binds to a linear epitope located in the cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)]

- 50–69: Used to test exposure of gp41 upon sCD4 binding [Klasse & Sattentau(1996)]
- 50–69: Binding of anti-gp120 MAbs IgG1b12 or 654–30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 50–69: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 50–69 maps to an immunodominant domain in gp41 – three groups of peptides were selected, one which seems most closely related to gp41 sequence peptide consensus is WGCxx(RK)(x n)LxC – the analogous gp41 sequence WGCSGKLIC is present in most M group clades, except D with a common L to H substitution [Boots (1997)]
- 50–69: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 – identifies non-contiguous W596-G597-C598 and C604-T605 as minimal epitope [Mitchell (1998)]
- 50–69: This antibody binds to acluster I epitope in rgp41, 567–647, and recognizes a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41 – this MAb doesn‘t react with either of the peptides N51 or C43 individually – MAbs 50–69 and 1367 had similar properties – MAb 50–69 bound the fusogenic form of the protein in liquid phase [Gorny & Zolla-Pazner(2000)]
- 50–69: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 50–69: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 50–69 bound the majority of isolates although binding was moderate to weak – specifies discontinuous binding site range as aa 579–613 [Nyambi (2000)]
- 50–69: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E – MAb 50–69 binding to infected cells is enhanced by sCD4, while 4E10 and Z13 binding is essentially unaltered [Zwick (2001b)]
- 50–69: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 50–69: NIH AIDS Research and Reference Reagent Program: 531

---

575 9–11 | gp160(579–604) | gp41(584–609) | RILAVERYLKDQQLLGIWGCS-GKLIC | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* protein *HIV component:* gp160

References: [Mani (1994)]

- 9–11: required the C-C disulfide bridge and loop formation, can bind simultaneously with 41–1 [Mani (1994)]

---

576 98–43 | gp160(579–604) | gp41(579–604 HXB2) | RILAVERYLKDQQLLGIWGCS-GKLIC | no HIV-1 infection | human(IgG2κ)

References: [Pinter (1989), Gorny (1989), Tyler (1990), Xu (1991)]

- 98–43: Reacts equally well with oligomer and monomer [Pinter (1989)]
- 98–43: Poor ADCC (in contrast to MAb 120–16, gp41(644–663)) [Tyler (1990)]
- 98–43: 579–604 binds in the immunodominant region [Xu (1991)]
- 98–43: NIH AIDS Research and Reference Reagent Program: 1241

---

577 41–1 (41.1) | gp160(579–608) | gp41(584–609) | RILAVERYLKDQQLLGIWGCS-GKLICTTAV | Vaccine | murine(IgG1κ)

Vaccine: *Vector/type:* protein *HIV component:* gp160

References: [Gosting (1987), Dalgleish (1988), Pincus (1991), Pincus & McClure(1993), Mani (1994), Pincus (1996), Pincus (1998)]

- 41–1: This antibody to gp41(584–609) [Mani (1994)] seems to have been named the same as a different MAb to gp41(735–752 IIIB) [Dalgleish (1988)]
- 41–1: Also called 41.1, although possibly not, the literature is confusing because two gp41 MAbs that bind to this region with similar names (dash versus period) are listed as murine and human
- 41–1: Broadly reactive [Gosting (1987)]
- 41–1: This antibody seems to have been named the same as a different MAb to gp41(735–752) [Dalgleish (1988)]
- 41–1: Efficacious as an immunotoxin when coupled to RAC – gave linear epitope as gp160 579–603 [Pincus (1991)]
- 41–1: Called 41.1, and described as a human MAb – cross-competes with 41.4 – sCD4 enhances the efficacy of immunotoxins *in vitro* 30-fold – MAb was coupled to ricin A chain (RAC) [Pincus & McClure(1993)]
- 41–1: Did not require the C-C disulfide bridge and loop formation, can bind simultaneously with 9–11 [Mani (1994)]
- 41–1: Called 41.1, and described as a human MAb, binding 579–604 – a panel of immunotoxins was generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]

---

578 41.4 | gp160(579–608) | gp41(584–609) | RILAVERYLKDQQLLGIWGCS-GKLICTTAV | ( )

Donor: Jan McClure, Bristol-Myers Squibb Pharmaceutical Res Inst, Seattle, WA
References: [Pincus & McClure(1993)]

- 41.4: Binds to peptide weakly, but to gp160 with higher affinity than 41.1, and cross-competes with 41.1 – probably conformational – MAb was coupled to ricin A chain (RAC) – sCD4 enhances the efficacy of immunotoxins *in vitro* 30-fold [Pincus & McClure(1993)]

---

579 Fab A1 | gp160(579–608) | gp41(584–609 LAI) | RILAVERYLKDQQLLGIWGCS-GKLICTTAV | no HIV-1 infection | human(IgG1κ)

References: [Binley (1996)]

- Fab A1: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

---

580 Fab A4 | gp160(579–608) | gp41(584–609 LAI) | RILAVERYLKDQQLLGIWGCS-GKLICTTAV | no HIV-1 infection | human(IgG1κ)

References: [Binley (1996)]

- Fab A4: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

581	Fab M12B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)

References: [Binley (1996)]
- Fab M12B: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

582	Fab M26B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)

References: [Binley (1996)]
- Fab M26B: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

583	Fab M8B	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)

References: [Binley (1996)]
- Fab M8B: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

584	Fab T2	gp160(579–608)	gp41(584–609 LAI)	RILAVERYLKDQQLLGIWGCS-GKLICTTAV	no HIV-1 infection	human(IgG1κ)

References: [Binley (1996)]
- Fab T2: Binds to cluster I region – competes with MAbs 240-D and 50–69 – conformation sensitive – variable regions sequenced [Binley (1996)]

585	86 (No. 86)	gp160(579–613)	gp41(586–620 IIIB)	RILAVERYLKDQQLLGIWGCS-GKLICTTAVPWNAS	no HIV-1 infection	human(IgG1κ)

Donor: Evan Hersh and Yoh-Ichi Matsumoto
References: [Sugano (1988), Robinson (1990b), Robinson (1990c), Pincus (1991), Moran (1993), Wisnewski (1996), Mitchell (1998)]
- 86: Reacts with gp41 and also reacted weakly with gp120 [Sugano (1988)]
- 86: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity in the presence of complement [Robinson (1990b)]
- 86: Peptide 586–620 blocks complement mediated ADE [Robinson (1990c)]
- 86: Poor immunotoxin activity when coupled to RAC – peptide binding stated to be aa 579–603 [Pincus (1991)]
- 86: Heavy (V HI) and light (V κI) chain sequenced – enhancing activity – similar germline sequence to MAb S1–1, but very different activity [Moran (1993)]
- 86: 86 is V H1 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]

- 86: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 86: NIH AIDS Research and Reference Reagent Program: 380

---

586 polyclonal | gp160(580–597) | gp41(584–602) | ILAVERYLKDQQLLGIWG | no | HIV-1 infection | human sera( )

References: [Petrov (1990)]

- Immunodominant and broadly reactive peptide [Petrov (1990)]

---

587 V10–9 | gp160(580–613) | gp41(586–620 IIIB) | ILAVERYLKDQQLLGIWGCSG-KLICTTAVPWNAS | no | HIV-1 infection | human(IgG1κ)

References: [Robinson (1990b), Robinson (1990c)]

- V10–9: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb 120–16 [Robinson (1990b)]
- V10–9: Peptide 586–620 blocks complement mediated ADE [Robinson (1990c)]

---

588 polyclonal | gp160(582–589) | gp41(589–596) | AVERYLKD | HIV-1 infection | human sera( )

References: [Klasse (1991)]

- Substitutions and deletions in peptide 583–599 were systematically studied – alterations in AVERYLKD abrogated the antigenicity of peptides with most of 14 human sera [Klasse (1991)]

---

589 polyclonal | gp160(584–604) | gp41(74–94) | ERYLKDQLLGIWGCSGKLIC | HIV-1 infection | human( )

References: [Shafferman (1989)]

- Immunogenic domain useful for diagnostics [Shafferman (1989)]

---

590 polyclonal | gp160(584–612) | gp41(587–617 BRU) | ERYLKDQQLLGIWGCSGKLIC-TTAVPWNA | no | HIV-1 infection | human( )

References: [Hernandez (2000)]

- Chimeric peptide combining two peptides gp160(495–516 and 584–612) served as a specific and broadly reactive antigen for diagnostic detection of HIV-1 [Hernandez (2000)]

---

591 2F11 | gp160(589–600) | gp41(589–600 HXB2) | DQQLLGIWGCSG | no | HIV-1 infection | human(IgG1)

References: [Eaton (1994)]

- 2F11: Enhances infectivity even in the absence of complement – does not mediate ADCC or neutralize virus [Eaton (1994)]

---

592 246-D (SZ-246.D) | gp160(590–597) | gp41(579–604 HXB2) | qqLLGIWg | no | HIV-1 infection | human(IgG1κ)

Ab type: cluster I **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU Med Center, NY, NY
References: [Xu (1991), Robinson (1991), Spear (1993), Eddleston (1993), Forthal (1995), Manca (1995), Saarloos (1995), Earl (1997), Gorny & Zolla-Pazner(2000), Nyambi (2000), Verrier (2001)]

- 246-D: Fine mapping indicates core is LLGI [Xu (1991)]
- 246-D: Did not mediate deposition of complement component C3 on HIV infected cells unless cells were pre-incubated with sCD4 [Spear (1993)]
- 246-D: No neutralizing activity, some enhancing activity [Robinson (1991)]
- 246-D: Called SZ-246.D [Eddleston (1993)]
- 246-D: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 246-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 246-D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed "Ab independent" in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos (1995)]
- 246-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 246-D: This antibody, along with murine MAb D61, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) [Earl (1997)]
- 246-D: Core epitope aa 591 to 597, a cluster I epitope that does not bind to either a peptide complex that approximates the core of the fusogenic form of gp41 or the individual peptides N51 and C43 that form this structure – MAbs 181-D and 246-D had similar properties [Gorny & Zolla-Pazner(2000)]
- 246-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to all 26 HIV-1 group M clades viruses tested and showed the strongest binding of all anti-Env MAbs tested, including the V3 and C5 region MAbs – notes core epitope as LLGI – no neutralizing activity was observed when 246-D was tested with five isolates [Nyambi (2000)]
- 246-D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 246-D: NIH AIDS Research and Reference Reagent Program: 1245

---

593 9G5A | gp160(591–594) | gp41(596–599 IIIB) | QLLG | | anti-idiotype | murine(IgM)

References: [Lopalco (1993), Beretta & Dalgleish(1994)]

- 9G5A: Anti-idiotype to gp120 C terminus (C5 region) MAb M38 [Lopalco (1993)]

---

594 181-D (SZ-181.D) | gp160(591–597) | gp41(591–597 HXB2) | qLLGIWg | no | HIV-1 infection | human(IgG2κ)

Ab type: cluster I **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Fontenot (1995), Gorny & Zolla-Pazner(2000), Nyambi (2000)]

- 181-D: Fine mapping indicates core is LLGIW [Xu (1991)]
- 181-D: No enhancing or neutralization activity [Robinson (1991)]
- 181-D: Called SZ-181.D [Eddleston (1993)]

- 181-D: No neutralizing, no ADCC, and no viral enhancing activity [Forthal (1995)]
- 181-D: Core epitope aa 591 to 597, a cluster I epitope that does not bind to either a peptide complex that approximates the core of the fusogenic form of gp41 or the individual peptides N51 and C43 that form this structure – MAbs 181-D and 246-D had similar properties [Gorny & Zolla-Pazner(2000)]
- 181-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 181-D bound the majority of isolates although binding was moderate to weak [Nyambi (2000)]

---

595 240-D (F240:) gp160(592–600) gp41(592–600 HXB2) LLGIWGCSG no HIV-1 infection human( )

Ab type: cluster I **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY
References: [Xu (1991), Robinson (1991), Spear (1993), Binley (1996), Wisnewski (1995), Wisnewski (1996), Mitchell (1998), Nyambi (2000)]

- 240-D: Fine mapping indicates core is IWG [Xu (1991)]
- 240-D: No neutralizing activity, some enhancing activity [Robinson (1991)]
- 240-D: Did not mediate deposition of complement component C3 on HIV infected cells [Spear (1993)]
- 240-D: Binds to a linear epitope located in the cluster I region – binding of 50–69 and 240-D inhibited by Fabs A1, A4, M8B, M26B, M12B and T2 [Binley (1996)]
- 240-D: Called F240: F240 in V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]
- 240-D: Mutations in BH10 gp160, W596Y and T605A, as well as deletions of 605–609 (TTAVP) and 597–609 (GCSGKLICTTAVP), abrogate binding of enhancing MAbs 86, 240D, 50–69, and 246-D – 5/6 enhancing MAbs identified to date bind to the immunodominant region 579–613 [Mitchell (1998)]
- 240-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 5 cluster I anti-gp41 MAbs which showed good cross clade reactivity – 246-D bound strongly or moderately to 24/26 HIV-1 group M clades viruses tested [Nyambi (2000)]
- 240-D: NIH AIDS Research and Reference Reagent Program: 1242

---

596 F240 gp160(592–606) gp41(592–606 BH10) LLGIWGCSGKLICTT no HIV-1 infection human(IgG1κ)

Ab type: cluster I **Donor:** L. Cavacina or M. Posner, Dept. of Med. Harvard Med. School, Boston MA, USA
References: [Cavacini (1998a), York (2001)]

- F240: Seems to be distinct from MAb 240-D, an antibody with a similar epitope in the immunodominant region of gp41 – dose-dependent reactivity with HIV isolates RF, SF2, IIIB, and MN was observed – F240 had no neutralizing activity and enhances infection in the presence of complement – reactivity of F240 is enhanced by preincubation of cells with sCD4 or anti-CD4BS MAb F105 – heavy and light chain variable domains were sequenced, and a strong homology to hu MAb 3D6 was observed, as 3D6 binds to the same epitope, these MAbs may define a human Ab clonotype [Cavacini (1998a)]

- F240: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]

---

597 D49 gp160(592–608) gp41(597–613) LLGIWGCSGKLICTTAV Vaccine murine( )

Vaccine: *Vector/type:* protein *HIV component:* dimeric Env

Ab type: cluster I **References:** [Earl (1994), Earl (1997)]

- D49: Binding maps to region 597–613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

---

598 D61 gp160(592–608) gp41(592–608 HXB2) LLGIWGCSGKLICTTAV Vaccine murine( )

Vaccine: *Vector/type:* protein *HIV component:* dimeric Env

Ab type: cluster I **References:** [Earl (1994), Richardson (1996), Weissenhorn (1996), Earl (1997)]

- D61: Linear gp41 epitope in the cluster I region – human sera blocked binding in oligomeric ELISA assay to a similar extent for gp41 MAbs D20, D43, D61, and T4 [Richardson (1996)]
- D61: Does not precipitate gp41(21–166), but due to a structural difference in the disulfide bonding region near the two cysteines – the authors propose that this region may change conformation during the activation of the membrane fusion state of the HIV-1 glycoprotein [Weissenhorn (1996)]
- D61: Binding maps to region 597–613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues – this antibody, along with human MAb 246-D, can be blocked by any of a group of 8 conformational MAbs (M10, D41, D54, T4, T6, T9, T10 and T35) – members of this competition group are blocked by sera from HIV-1+ individuals [Earl (1997)]

---

599 T32 gp160(592–608) gp41(597–613) LLGIWGCSGKLICTTAV Vaccine murine( )

Vaccine: *Vector/type:* tetrameric Env *HIV component:* Env

Ab type: cluster I **References:** [Earl (1994), Earl (1997)]

- T32: Binding maps to region 597–613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

---

600 T34 gp160(592–608) gp41(597–613) LLGIWGCSGKLICTTAV Vaccine murine( )

Vaccine: *Vector/type:* tetrameric Env *HIV component:* Env

Ab type: cluster I **References:** [Earl (1994), Earl (1997)]

- T34: Binding maps to region 597–613: WGCSGKLICTTAVPWNA – immunodominant region containing two Cys residues [Earl (1997)]

601	115.8	gp160(593–604)	gp41(598–609)	LGLIWGCSGKLIC	Vaccine	murine(IgM)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 115.8: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor reactivity with CSGKLIC – reacts well with longer HIV-2 peptide NSWGCAFRQVC as well as CAFRQVC – disulfide bond between cysteines required [Oldstone (1991)]

602	M-1	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1,IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-1: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]

603	M-11	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-11: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]

604	M-13	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-13: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]

605	M-2	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-2: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]

606	M-22	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-22: Strongest reaction of 12 anti-HIV-1 gp41 MAbs to a cellular 43-kd protein found in rat and human astrocytes [Yamada (1991)]

607	M-24	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Yamada (1991)]

- M-24: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]

608	M-25	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-25: Reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]				
609	M-28	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-28: Strongly reacted with a cellular 43-kd protein found in rat and human astrocytes as well as with gp41 [Yamada (1991)]				
610	M-29	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-29: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]				
611	M-36	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG1)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-36: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]				
612	M-4	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-4: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]				
613	M-6	gp160(593–604)	gp41(598–609)	LGIWGCSGKLIC	Vaccine	murine(IgG2b)
	Vaccine:	*Vector/type:* peptide	*HIV component:* gp41			
		References: [Yamada (1991)]				
		• M-6: Unlike M-22, did not react to 43-kd protein found in rat and human astrocytes [Yamada (1991)]				
614	polyclonal α(598–609)	gp160(594–601)	gp41(598–609)	GIWGCSGK	HIV-1 infection	human( )
		References: [Poumbourios (1992)]				
		• α(598–609): Affinity purified from HIV-1+ plasma – immunodominant region, binds oligomer and monomer [Poumbourios (1992)]				

615	1B8.env	gp160(594–604)	gp41(594–605 HXB2)	GIWGCSGKLIC	no	HIV-1 infection	human(IgG2λ)

References: [Banapour (1987)]

- 1B8.env: Highly conserved epitope recognized by the majority of HIV-1 infected people [Banapour (1987)]

616	polyclonal	gp160(594–609)	gp41(601–616)	GIWGCSGKLICTTAVP	no	HIV-1 infection	human sera( )

References: [Petrov (1990)]

- Immunodominant and broadly reactive peptide [Petrov (1990)]

617	clone 3	gp160(597–606)	gp41(597–606)	GCSGKLICTT	L	HIV-1 infection	human(IgG1)

References: [Cotropia (1992), Cotropia (1996)]

- clone 3: Core binding domain gcsgkLIC – lack of serological activity to this region correlates with rapid progression in infants ([Broliden1989]) [Cotropia (1992)]
- clone 3: Inhibits replication of three diverse HIV-1 laboratory strains, as well as an AZT-resistant isolate [Cotropia (1996)]

618	4	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 4: There is another MAb with this ID that reacts with integrase [Oldstone (1991), Bizub-Bender (1994)]
- 4: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with longer HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]

619	41–6	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 41–6: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – slightly more reactive with LGLIWGCSGKLIC and HIV-2 form NSWGCAFRQVC – disulfide bond between cysteines required [Oldstone (1991)]

620	41–7	gp160(598–604)	gp41(605–611)	CSGKLIC	no	HIV-1 infection	human(IgG1κ)

References: [Bugge (1990)]

- 41–7: Sera from 6/6 HIV-1 positive, but no HIV-2 positive individuals, interfered with 41–7 binding [Bugge (1990)]

621	68.1	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgM)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 68.1: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]

622	68.11	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	murine(IgM)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 68.11: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – cross-reactive with HIV-2 peptide CAFRQVC – more reactive with longer HIV-1 peptide LGLIWGCSGKLIC and HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]

623	75	gp160(598–604)	gp41(598–609)	CSGKLIC		Vaccine	rat(IgG)

Vaccine: *Vector/type:* peptide *HIV component:* gp41

References: [Oldstone (1991)]

- 75: Stimulated by immunization with the peptide: LGLIWGCSGKLIC (aa 598–609) – poor cross-reactivity with HIV-2 peptide CAFRQVC – more reactive with longer HIV-2 peptide NSWGCAFRQVC [Oldstone (1991)]

624	105–732	gp160(599–606)	gp41(601–608 HAM112, O group)	KGRLICYT		Vaccine	murine(IgG2bκ)

Vaccine: *Vector/type:* recombinant protein *Strain:* HAM112 (group O) *HIV component:* gp160

References: [Scheffel (1999)]

- 105–732: Overlapping peptides based on group O HAM112 Env were tested for MAb reactivity – MAb 105–732 bound to two overlapping peptides [Scheffel (1999)]

625	3D6 (IAM 41–3D6)	gp160(599–613)	gp41(604–617 BH10)	SGKLICTTAVPWNAS	no	HIV-1 infection	human(IgG1κ)

Ab type: immunodominant region **Donor:** H. Katinger, Inst. Appl. Microbiol., Vienna, Austria and Viral Testing Systems, Houston, TX

References: [Felgenhauer (1990), He (1992), Chen (1994b), Sattentau (1995), Stigler (1995), Wisnewski (1996), Kunert (1998), Cavacini (1998b), Cavacini (1998a), Cavacini (1999)]

- 3D6: Sequence of cDNA encoding V- regions [Felgenhauer (1990)]
- 3D6: Fab fragment crystal structure [He (1992)]
- 3D6: This MAb binds to HIV gp41, and to a 43 kd protein found in human T, B and monocyte cell lines, proposed molecular mimicry [Chen (1994b)]
- 3D6: Called IAM 41–3D6: binding increased after pretreatment of infected cells with sCD4 – binding domain overlaps site that is critical for gp120-gp41 association [Sattentau (1995)]
- 3D6: Optimum peptide for binding 3D6 Fab was CSGKLICTTAVPW [Stigler (1995)]
- 3D6: 3D6 is V H3 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]
- 3D6: The complete V, J and D(H) domain was sequenced – in contrast the sequences of five neutralizing MAbs, 3D6 had very little somatic mutation, with homologies of 97–98% relative to germline genes [Kunert (1998)]
- 3D6: Binds to the immunodominant region of gp41 – a strong homology between heavy variable domains of hu MAb 3D6 and MAb F20 was observed, these MAbs may define a human Ab clonotype [Cavacini (1998a)]

- 3D6: Cavacini *et al*. note that both MAbs F223 and 3D6 are anti-HIV-1 Env MAbs that have an autoimmune response and that both uses VH3 germline genes [Cavacini (1999)]

---

626 F172-D8 (F172-D8, scFvD8) | gp160(604–615) | gp41(609–620) | CTTAVPWNASWS? | | | human( )

References: [Legastelois & Desgranges(2000)]

- F172-D8: As an approach to intercellular immunization using a single-chain variable fragment, scFvD8 was constructed based on the MAb F172-D8, directed at a loop in gp41 between the two heptad repeat regions – intracellular scFvD8 expression decreased gp160 expression and a scFvD8 transfected cell line did not support infection by HIV-1 Ba-L or primary isolates [Legastelois & Desgranges(2000)]

---

627 D50 | gp160(632–655) | gp41(642–665) | | | Vaccine | murine( )

Vaccine: *Vector/type:* protein *HIV component:* dimeric Env

Ab type: cluster II **References:** [Earl (1994), Binley (1996), Richardson (1996), Earl (1997), Srivastava (2002)]

- D50: Thought to be a discontinuous epitope recognizing residues between 649–668 – designated cluster II – Fabs D5, D11, G1, T3, M12, M15, S6, S8, S9, S10 block binding [Binley (1996)]
- D50: Richardson suggests this is a linear gp41 epitope [Richardson (1996)]
- D50: Found to bind to a linear peptide, between Env amino acids 642–655 – can be blocked by the conformation dependent MAbs D16, D17, D31, D36, D37, D40, D44, D55, D59, T37, and T45 – the region is in the immunogenic cluster two region – reactive with 9/10 HIV-1 strains tested, all except HIV-1 ADA, in which the change E659D and E662A may result in the loss of binding (ELLE to DLLA) [Earl (1997)]
- D50: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – D50 was used to capture the o-gp140 for ELISA to test the antigencity of o-gp140 using a panel of well characterized MAbs [Srivastava (2002)]

---

628 5–21-3 | gp160(642–665) | gp41(642–665 HXB2) | IHSLIEESQNQQEKNEQELLE-LDK | | Vaccine | murine( )

Vaccine: *Vector/type:* recombinant protein *HIV component:* gp41

References: [Hunt (1990), Scheffel (1999)]

- 5–21-3: Recognizes a contiguous, conformation-dependent epitope in a hydrophilic region [Hunt (1990)]
- 5–21-3: Binds group M gp41, used as a control in a study of group O MAbs [Scheffel (1999)]

---

629 120–16 (SZ-120.16) | gp160(644–663) | gp41(644–663 HXB2) | SLIEESQNQQEKNEQELLEL | no | HIV-1 infection | human(IgG2κ)

References: [Andris (1992), Robinson (1990b), Tyler (1990), Xu (1991), Robinson (1991), Eddleston (1993), Forthal (1995), Wisnewski (1996)]

- 120–16: Antibody dependent enhancement (ADE) of HIV-1 IIIB infectivity, synergistically enhanced by MAb V10–9 [Robinson (1990b)]
- 120–16: Potent ADCC (in contrast to MAb 98–43, gp41(579–604)) [Tyler (1990)]

- 120–16: Less reactive region than AVERY region – most Abs involving this region bound conformational epitopes, this was the only linear one [Xu (1991)]
- 120–16: Synergizes with huMAb 50–69 *in vitro* to enhance HIV-1 infection [Robinson (1991)]
- 120–16: Called SZ-120.16 [Eddleston (1993)]
- 120–16: No neutralizing activity, both ADCC and viral enhancing activity [Forthal (1995)]
- 120–16: 120–16 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]

---

630 98–6 (SZ-98.6) | gp160(dis 644–663) | gp41(dis 644–663 HXB2) | SLIEESQNQQEKNEQELLEL | no | HIV-1 infection | human(IgG2κ)

Ab type: α-helical bundle **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY

References: [Pinter (1989), Gorny (1989), Till (1989), Robinson (1990b), Tyler (1990), Andris (1992), Sattentau & Moore(1991), Robinson (1991), Xu (1991), Eddleston (1993), Spear (1993), Tani (1994), Laal (1994), Chen (1995), Forthal (1995), Manca (1995), Sattentau (1995), Wisnewski (1996), Nyambi (1998), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000), Taniguchi (2000), Verrier (2001)]

- 98–6: Reacts preferentially with gp160 oligomer, compared to gp41 monomer [Pinter (1989)]
- 98–6: Kills HIV-infected cells when coupled to deglycosylated ricin A chain [Gorny (1989)]
- 98–6: Toxic to HIV-infected T cells (H9) and monocytes (U937) when coupled to deglycosylated A chain of ricin [Till (1989)]
- 98–6: No neutralizing or enhancing activity for HIV-1 IIIB [Robinson (1990b)]
- 98–6: Serves as target for antibody-dependent cellular cytotoxicity, ADCC [Tyler (1990)]
- 98–6: Two fold increase in binding to gp120 in the presence of bound sCD4 [Sattentau & Moore(1991)]
- 98–6: No neutralizing or enhancing activity [Robinson (1991)]
- 98–6: Appeared to be specific for a conformational or discontinuous epitope [Xu (1991)]
- 98–6: Called SZ-98.6 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 167–7 and ND-15G1 [Eddleston (1993)]
- 98–6: Did not mediate deposition of complement component C3 on HIV infected cells, binding enhanced by sCD4 [Spear (1993)]
- 98–6: This MAb was expressed as a surface anti-gp41 monoclonal antibody receptor for gp41 on a CD4-negative B-cell line. Transfected cells could bind HIV Envelope, but could not be infected by HIV-1. When CD4 delivered by retroviral constructs was expressed on these cells, they acquired the ability to replicate HIV-1, and sIg/gp41 specifically enhanced viral replication [Tani (1994)]
- 98–6: Epitope described as cluster II, 644–663, conformational – does not neutralize IIIB or synergize neutralization by anti-V3 MAb 447-52D or by CD4 BS MAbs [Laal (1994)]
- 98–6: One of several anti-gp41 MAbs that bind to a gp41-maltose binding fusion protein designed to study the leucine zipper domain of gp41, showing that the construct has retained aspects of normal gp41 conformation [Chen (1995)]
- 98–6: No neutralizing activity, positive ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 98–6: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 98–6: Preferentially recognizes oligomeric form of gp41 – enhanced binding to HIV-1 infected cells at 37 degrees relative to 4 degrees – addition of sCD4 enhances binding [Sattentau (1995)]
- 98–6: 98–6 is V H4 – V-region heavy chain usage was examined and a bias of enhanced V H1 and V H4, and reduced V H3, was noted among HIV infected individuals [Wisnewski (1996)]

- 98–6: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – anti-gp41 Abs 98–6, 1367 and 1342 were not able to bind detectably with any of the viruses from any clade [Nyambi (1998)]
- 98–6: 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and the binding of 98–6 is not inhibited by N51 [Gorny & Zolla-Pazner(2000)]
- 98–6: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 98–6: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs – no neutralizing activity was observed when tested against 5 isolates, but 98–6 did not bind to these isolates [Nyambi (2000)]
- 98–6: The fusogenic form of gp41 is recognized by 98–6, and the epitope is a conformational epitope formed by the interaction of two regions of gp41 which form an α-helical bundle [Taniguchi (2000)]
- 98–6: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 98–6: NIH AIDS Research and Reference Reagent Program: 1240

---

631 167–7 (SZ-167.7) | gp160(644–663) | gp41(644–663) | SLIEESQNQQEKNEQELLEL | | HIV-1 infection | human(IgG2λ)

Ab type: cluster II **References:** [Xu (1991), Eddleston (1993)]

- 167–7: Specific for a conformational epitope [Xu (1991)]
- 167–7: Called SZ-167.7 – binds to a conformational domain within aa 644–663 of gp41, and reacts with astrocytes, as do 98–6 and ND-15G1 [Eddleston (1993)]

---

632 167-D | gp160(644–663) | gp41(644–663 HXB2) | SLIEESQNQQEKNEQELLEL | no | HIV-1 infection | human(IgG1λ)

Ab type: cluster II **Donor:** Susan Zolla-Pazner (Zollas01@mcrcr6.med.nyu), NYU, NY
References: [Spear (1993), Forthal (1995), Gorny & Zolla-Pazner(2000), Gorny (2000), Nyambi (2000)]

- 167-D: Did not mediate deposition of complement component C3 on HIV infected cells – complement mediated virolysis of MN and IIIB in the presence of sCD4 [Spear (1993)]
- 167-D: No neutralizing activity, no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 167-D: Virions complexed to gp41 Ab facilitate presentation of p66 RT epitopes to Th cells [Manca (1995)]
- 167-D: This cluster II MAb binds to a conformational epitope in the region 644–663 – like most cluster II MAbs (126-6, 167-D, 1281, 1342, and 1379 all reacted similarly) it binds to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, but not to C43 nor to N51 alone [Gorny & Zolla-Pazner(2000)]

- 167-D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, but gp41 MAb 50–69 bound with a 5 fold preference for the oligomer, while other gp41 MAbs (1367, 98–6, 167-D, 1281, 1342, and 1379) did not show a preference [Gorny (2000)]
- 167-D: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs [Nyambi (2000)]

---

633 ND-15G1 | gp160(644–663) | gp41(644–663 HXB2) | SLIEESQNQQEKNEQELLEL | | HIV-1 infection | human(IgG1κ)

Ab type: cluster II **References:** [Eddleston (1993)]

- ND-15G1: Mapped to the conformational epitope within aa 644–663, and reacts with astrocytes, as do 98–6 and 167–7 [Eddleston (1993)]

---

634 2F5 (IAM 2F5, IAM-41–2F5, IAM2F5, c2F5) | gp160(dis 656–671) | gp41(dis 662–667 BH10) | NEQELLELDKWASLWN | L P | HIV-1 infection | human(IgG3κ)

Ab type: adjacent to cluster II **Donor:** Hermann Katinger, U. of Bodenkultur, or Polymun Scientific Inc., Vienna, Austria, or Viral Testing Systems Corp., Houson TX

References: [Buchacher (1992), Muster (1993), Allaway (1993), Klasse (1993a), Purtscher (1994), Laal (1994), Buchacher (1994), D'Souza (1994), Conley (1994b), Thali (1994), Chen (1994b), Muster (1994), Beretta & Dalgleish(1994), D'Souza (1995), Trkola (1995), Sattentau (1995), Moore & Ho(1995), Neurath (1995), Kessler 2nd (1995), Calarota (1996), McKeating(1996), Poignard (1996b), Sattentau(1996), Conley (1996), Pincus (1996), McKeating (1996), Stoiber (1996), Purtscher (1996), Schutten (1997), D'Souza (1997), Mo (1997), Li (1997), Kessler II (1997), Moore & Trkola(1997), Mascola (1997), Stamatatos (1997), Turbica (1997), Ugolini (1997), Burton & Montefiori(1997), Earl (1997), Gorny (1997), Andrus (1998), Mondor (1998), Connor (1998), Parren (1998a), Yang (1998), Trkola (1998), Fouts (1998), Ernst (1998), Takefman (1998), Li (1998), Jiang (1998), Parren (1998b), Geffin (1998), Kunert (1998), Frankel (1998), Montefiori & Evans(1999), Poignard (1999), Beddows (1999), Muhlbacher (1999), Parren (1999), Mascola (1999), Mascola (2000), Baba (2000), Gorny & Zolla-Pazner(2000), Kunert (2000), Liao (2000), Lu (2000b), Lu (2000a), Nyambi (2000), Park (2000), Xiao (2000c), Dong (2001), Kolchinsky (2001), Tumanova (2001), York (2001), Zwick (2001b), Zwick (2001c), Mascola & Nabel(2001), Barnett (2001), Moore (2001), Zeder-Lutz (2001), Parker (2001), Spenlehauer (2001), Verrier (2001), Stiegler (2001), Hofmann-Lehmann (2001), Xu (2001), Sanhadji (2000), Coeffier (2000), Armbruster (2002), Srivastava (2002)]

- 2F5: DKWA defined as the core sequence – highly conserved epitope neutralizing MAb [Buchacher (1992), Muster (1993)]
- 2F5: Synergy with combinations of CD4-based molecules in inhibition of HIV-1 Env mediated cell fusion [Allaway (1993)]
- 2F5: Called IAM-41–2F5 – reports MAb to be IgG1 – the gp41 mutation 582(Ala to Thr) results in conformational changes in gp120 that confer neutralization resistance to conformationally sensitive neutralizing MAbs – neutralization efficiency of 2F5 is not affected [Klasse (1993a)]

- 2F5: Broadly reactive neutralizing activity, core epitope, ELDKWA, is relatively conserved – neutralized 2 primary isolates [Purtscher (1994)]
- 2F5: Failed to show synergy with anti-CD4 binding site IIIB neutralizing antibodies [Laal (1994)]
- 2F5: MAb generated by electrofusion of PBL from HIV-1 positive volunteers with CB-F7 cells [Buchacher (1994)]
- 2F5: Included in a multi-lab study for antibody characterization binding and neutralization assay comparison [D'Souza (1994)]
- 2F5: Called IAM-41–2F5 – neutralized lab and primary isolates – $t_{1/2}$ dissociation 122 min for the peptide, and 156 min for gp41 – core D(K/R)W – Ab resistant isolate had the sequence KLDNWA [Conley (1994b)]
- 2F5: gp41 mutation (582 A/T) that reduces neutralization of anti-CD4 binding site MAbs does not alter 2F5's ability to neutralize [Thali (1994)]
- 2F5: 2F5 core epitope ELDKWA inserted into an immunogenic loop in influenza virus hemagglutinin can elicit IIIB, MN and RF neutralizing sera in immunized mice [Muster (1994)]
- 2F5: Found to neutralize MN, JRCSF, and two B subtype primary isolates, but not a D subtype primary isolate, by most labs in a multi-laboratory study involving 11 labs [D'Souza (1995)]
- 2F5: Cross-clade primary virus neutralizing activity – LDKW defined as the core epitope [Trkola (1995)]
- 2F5: Called IAM 41–2F5 – exposed in the presence of gp120 on the cell surface, while most of gp41 is masked – binds proximal to transmembrane region [Sattentau (1995)]
- 2F5: Review: binds to the only generally accepted strong neutralizing epitope outside of gp120, one of only 3 MAbs with strong broad activity against primary viruses, the others are 2G12 and IgG1b12 – unique member of epitope cluster [Moore & Ho(1995)] and John Moore, per comm 1996
- 2F5: MAb binding decreases the accessibility or alters the conformation of the gp41 fusion domain and of gp120 domains, including the binding site for the CD4 cell receptor [Neurath (1995)]
- 2F5: Broad cross-clade neutralization of primary isolates – additive neutralization in combination with anti-CD4BS MAb IgG1b12 (Called BM12) [Kessler 2nd (1995)]
- 2F5: Only 4/20 Argentinian and 3/43 Swedish HIV+ sera reacted with LLELDKWASL – sera reacting with peptides that contained ELDKWA tended to have high neutralization titers – the region carboxyl terminal to EDLKWA was found to be more important for polyclonal sera AB binding, 670–675 WNWFDI – 2F5 bound most strongly to the peptide QELLELDKWA [Calarota (1996)]
- 2F5: ELDKWAS is in a gp41 binding region for the negative regulator of complement factor H (CFH) – Abs to HIV generally do not cause efficient complement-mediated lysis, but binding of 2F5 can interfere with CHF binding, facilitating HIV destruction by complement [Stoiber (1996)]
- 2F5: Primary isolates from clade A, B, and E are neutralized by 2F5 – neutralization requires the LDKW motif – neutralization resistant isolates or 2F5 selected variants all had substitutions in the D or K [Purtscher (1996)]
- 2F5: Neutralizes HXB2, primary isolates, and chimeric virus with gp120 from primary isolates in an HXB2 background [McKeating (1996)]
- 2F5: Review: one of three MAbs (IgG1b12, 2G12, and 2F5) generally accepted as having significant potency against primary isolates [Poignard (1996b)]
- 2F5: Review: only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]

- 2F5: 2F5 was infused into two chimpanzees which were then given an intravenous challenge with a primary HIV-1 isolate – both became infected, but with delayed detection and prolonged decrease in viral load relative to controls, indicating that preexisting, neutralizing antibodies (passively administered or actively elicited) affect the course of acute-phase virus replication and can be influential after the Ab can no longer be detected in the peripheral circulation [Conley (1996)]
- 2F5: A panel of immunotoxins were generated by linking Env MAbs to ricin A – immunotoxins mediated cell killing, but killing was not directly proportional to binding [Pincus (1996)]
- 2F5: Called IAM 2F5 – antibody mediated enhancement or inhibition seemed to be determined by isolate rather than antibody specificity – in this study, only 2F5 inhibited the entry of all the viruses studied, irrespective of their phenotype, and directly proportional to its affinity to monomeric HIV-1 gp160 [Schutten (1997)]
- 2F5: Of three neutralizing MAbs (257-D, IgG1b12, and 2F5), 2F5 was the only one to inhibit the entry of all viruses studied, both SI and NSI, with a potency proportional to its affinity for monomeric gp126 [Schutten (1997)]
- 2F5: In a multilab evaluation of monoclonal antibodies, only IgG1b12, 2G12, and 2F5 could neutralize at least half of the 9 primary test isolates at a concentration of < 25 μg per ml for 90% viral inhibition – the isolates with no 2F5 neutralizing susceptibility had the sequences ALGQWA or ELDTWA instead of EDLKWA – 7/9 primary isolates were neutralized, and ALDKWQ and ALDKWA were susceptible to neutralization [D'Souza (1997)]
- 2F5: A JRCSF variant that was selected for IgG1b12 resistance remained sensitive to MAbs 2G12 and 2F5, for combination therapy [Mo (1997)]
- 2F5: One of 14 human MAbs tested for ability to neutralize a chimeric SHIV-vpu+, which expressed HIV-1 IIIB Env – strong neutralizer of SHIV-vpu+ – all Ab combinations tested showed synergistic neutralization – 2F5 has synergistic response with MAbs 694/98-D (anti-V3), 2G12, b12, and F105 [Li (1997)]
- 2F5: IgG1b12 was more potent with greater breadth than MAb 2F5 in an infection reduction assay including 35 primary isolates [Kessler II (1997)]
- 2F5: Review: MABs 2F5, 2G12 and IgG1b12 have potential for use in combination with CD4-IgG2 as an immunotherapeutic or immunoprophylactic – homologous MAbs to these are rare in humans and vaccine strategies should consider including constructs that may enhance exposure of these MAbs‘ epitopes [Moore & Trkola(1997)]
- 2F5: Binding of anti-gp120 MAbs IgG1b12 or 654–30D does not mediate significant exposure of the gp41 epitopes for MAbs 2F5 and 50–69 [Stamatatos (1997)]
- 2F5: Using concentrations of Abs achievable *in vivo*, the triple combination of 2F5, 2G12 and HIVIG was found to be synergistic to have the greatest breadth and magnitude of response against 15 clade B primary isolates [Mascola (1997)]
- 2F5: Used to standardize polyclonal response to CD4 BS [Turbica (1997)]
- 2F5: The only MAb out of a large panel to show no correlation between Viral binding inhibition and neutralization [Ugolini (1997)]
- 2F5: This review summarizes results about 2F5: it binds extracellularly, near the transmembrane domain, it is the only gp41 MAb that is neutralizing, it reacts with many non-B clade viruses and has a paradoxically weak binding to virus, given the neutralizing titers [Burton & Montefiori(1997)]
- 2F5: Post-exposure prophylaxis was effective when MAb 694/98-D was delivered 15 min post-exposure to HIV-1 LAI in hu-PBL-SCID mice, but declined to 50% if delivered 60 min post-exposure, and similar time constraints have been observed for HIVIG, 2F5 and 2G12, in contrast to MAb BAT123 that could protect delivered 4 hours post infection [Andrus (1998)]

- 2F5: This MAb and the results of [Ugolini (1997)] are discussed – the authors propose that an Ab bound to gp41 would typically project less from the surface of the virion and so be unable to interfere with attachment [Parren (1998a)]
- 2F5: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- 2F5: A neutralization assay was developed based on hemi-nested PCR amplification of the LTR (HNPCR) – LTR-HNPCR consistently revealed HIV DNA and was shown to be a rapid, specific and reliable neutralization assay based on tests with 6 MAbs and 5 isolates [Yang (1998)]
- 2F5: A wide range of neutralizing titers was observed that was independent of co-receptor usage – 2F5 was the most potent of the MAbs tested [Trkola (1998)]
- 2F5: Points out that 2G12 and 2F5, potent neutralizing antibodies, were identified by screening for cell surface (oligomeric Envelope) reactivity [Fouts (1998)]
- 2F5: The ELDKWA core epitope was inserted into the antigenic site B of influenza hemagglutinin and expressed on baculovirus infected insect cells, flanked by 3 additional random amino acids, xELDKWAxx – FACS was used to isolate the clone that displayed the epitope with the most markedly increased binding capacity for 2F5, to identify particularly specific immunogenic constructs – PELDKWAPP was a high affinity form selected by FACS [Ernst (1998)]
- 2F5: Induces complement-mediated lysis in MN but not primary isolates – primary isolates are refractive to CML [Takefman (1998)]
- 2F5: Neutralization synergy was observed when the MAbs 694/98-D (V3), 2F5 (gp41), and 2G12 (gp120 discontinuous) were used in combination, and even greater neutralizing potential was seen with the addition of a fourth MAb, F105 (CD4 BS) [Li (1998)]
- 2F5: Used as a control in the study of anti-gp41 MAb NC-1 – 2F5 does not react with HIV-2 gp41 or gp160 [Jiang (1998)]
- 2F5: MAbs 2G12, 2F5 and b12 are broadly neutralizing, as are some human polyconal sera, but this paper describes a set of primary isolates that are resistant to all three MAbs and 2 broadly neutralizing sera – results indicate that resistance levels of pediatric isolates might be higher than adult isolates – resistance in general did not seem to be conferred by a loss of binding affinity for gp120 or gp41, rather by a more global perturbation of oligomeric Envelope [Parren (1998b)]
- 2F5: The natural immune response to the core epitope of 2F5, ELDKWA, was studied in perinatally infected children and levels of reactivity to this epitope were correlated with absolute CD4 numbers over time and health status – 3/10 children who had no antibody reactivity to ELDKWA had substitutions in the epitope (ALDKWA, ELDQWA, and KLDKWA) – 2F5 competed with the ELDKWA-reactive sera depending on the serum titer [Geffin (1998)]
- 2F5: The complete V, J and D(H) domain was sequenced – unlike non-neutralizing anti-gp41 MAb 3D6, five neutralizing MAbs (2F5, 2G12, 1B1, 1F7, and 3D5) showed extensive somatic mutations giving evidence of persistent antigenic pressure over long periods – in contrast to Geffin98, where multiple pediatric sera were found to compete with 2F5, cross-competition was noted to be very rare in sera from HIV+ adults – Kunert *et al*. propose that because there is a binding site of human complement factor H which overlaps the 2F5 binding site, it may generally be masked from the immune system – 2F5 also has a remarkably long CDR3 loop of 22 amino acids, and this region could not be readily assigned to any described D(H) fragment, leading to the suggestion of recombination of two fragments from novel regions [Kunert (1998)]
- 2F5: Infection of dendritic cells cultured from CD14+ blood cells or from cadaveric human skin was blocked by neutraling MAbs IgG1b12, or 2F5 and 2G12 delivered together, but not by control non-neutralizing anti-gp120 MAb 4.8D, indicating that NAbs could interrupt early mucosal transmission events [Frankel (1998)]

- 2F5: rgp120 derived from a R5X4 subtype B virus was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs [Beddows (1999)]
- 2F5: A meeting summary presented results regarding neutralization –MAbs 2G12 and 2F5 tested for their ability to neutralize primary isolate infection of genetically engineered cell lines (cMAGI and others, presented by T. Matthews, A. Trkola, J. Bradac) – an advantage of such cells lines over PBMCs is that markers (X-Gal) can be added for staining to simplify the assay – the consensus of the meeting was that these engineered cell lines did not improve the sensitivity of detection of primary isolate neutralization – D. Burton and J. Mascola presented results concerning passive immunization and protection of hu-PBL-SCID mice and macaques, respectively, and both found combinations of MAbs that were able to achieve 99% neutralization *in vitro* corresponded to efficacy *in vivo* [Montefiori & Evans(1999)]
- 2F5: Hu-PBL-SCID mice were infected with HIV-1s JRCSF and SF162 to study the effect of NAbs on an established infection – no significant differences in the initial rate of decrease in viral load or the plateau levels of viral RNA between the b12 treated and control mice were seen – in most of the Ab treated mice b12 escape mutants were observed with varying patterns of mutations – a combination of b12, 2G12 and 2F5 protected 1/3 mice, and an isolate from one of the other two was resistant to neutralization by all three MAbs [Poignard (1999)]
- 2F5: In a study of 116 HIV-1+ individuals, Ab reactivity to a peptide encompassing the ELDKWA peptide decreased in CDC stage C patients compared with stage A patients, and longitudinal studies showed a decline in 6/8 patients, while overall Ab reactivity to rec soluble gp160 stayed constant [Muhlbacher (1999)]
- 2F5: Review of the neutralizing Ab response to HIV-1 [Parren (1999)]
- 2F5: Combinations of HIVIG, 2F5, 2G12 were administered in passive-transfer experiments 24 hours prior to challenge with pathogenic SHIV 89.6PD – 3/6 animals given HIVIG/2F5/2G12 were completely protected, the others had reduced viremia and normal CD4 counts – 1/3 monkeys given 2F5/2G12 showed transient infection, the other two had reduced viral load – all monkeys that received HIVIG, 2F5, or 2G12 alone became infected and developed high-level plasma viremia, although animals that got HIVIG or 2G12 had a less profound CD4 T cell decline [Mascola (1999)]
- 2F5: Because HIV-1 is most often transmitted across mucosal surfaces, the ability of passive transfer of infused HIVIG/2F5/2G12 to protect against mucosal exposure of macaques to pathogenic SHIV 89.6PD was studied – HIVIG/2F5/2G12 protected 4/5 animals against vaginal challenge, 2F5/2G12 combined protected 2/5 animals, and 2G12 alone protected 2/4 animals – in contrast, Mascola and co-workers had previously shown single MAbs could not protect against intervenous challenge – Ab treated animals that got infected through vaginal innoculation had low viral loads and only modest declines in CD4 counts – the infused Abs were detected in the nasal, vaginal, and oral mucosa [Mascola (2000)]
- 2F5: Paper uses IgG1 form of 2F5 – a triple combination of 2F5, F105 and 2G12 effectively neutralized perinatal infection of macaque infants when challenged with SHIV-vpu+ – the plasma half-life was 4.2 ± 0.8 days [Baba (2000)]
- 2F5: MAbs 98–6 and 2F5 both bind to a peptide N51-C43 complex trimer of heterodimers that approximates the core of the fusogenic form of gp41, and to C43 alone but not to N51 alone – 98–6 and 2F5 have comparable affinities for C43, but 98–6 has a higher affinity for the complex and 2F5 may bind to an epitope of C43 that is directly involved with complex formation –and IgG1 rec form of the Ab was used in this study [Gorny & Zolla-Pazner(2000)]
- 2F5: 2F5 is a candidate for immunotherapy, but generally IgG1 has a longer half-life in humans than IgG3, so the isotype was switched – rec CHO-derived MAb 2F5 IgG1kappa and hybridoma-derived MAb 2F5 IgG3kappa displayed identical specificity, *in vitro* function, and epitope (ELDKWA) – it remains to be determined if isotype switching will prolong β-clearance [Kunert (2000)]

- 2F5: Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response [Liao (2000)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000b)]
- 2F5: ELDKWA peptide vaccine study [Lu (2000a)]
- 2F5: 26 HIV-1 group M isolates (clades A to H) were tested for binding to 47 MAbs, including 6 cluster II anti-gp41 MAbs – of these 2F5, 167-D, 126-6, and 1281 bound across clades, but usually weakly, while 98–6 and 1342 had poor cross reactivity – Clade D isolates bound most consistently to cluster II MAbs [Nyambi (2000)]
- 2F5: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i gp120 specific MAbs are 20–100 fold more efficient at neutralizing the sensitive form – gp41 MAbs bind less, and 2F5 behaves the opposite of gp120 MAbs in that it neutralizes the "sensitive" form less efficiently [Park (2000)]
- 2F5: Mutations in two glcosylation sites in the V2 region of HIV-1 ADA at positions 190 and 197 (187 DNTSYRLINCNTS 199) cause the virus to become CD4-independent and able to enter cells through CCR5 alone – these same mutations tended to increase the neutralization sensitivity of the virus, including to antibody 2F5 [Kolchinsky (2001)]
- 2F5: A peptide called 5-Helix was designed that binds to the C-peptide region of gp41 – 5-Helix is a potent inhibitor of HIV-1 entry that binds immediately COOH-terminal to the C-peptide region targeted by 5-Helix – the conformation of the bound 2F5 epitope is a hairpin turn [Root2001a]
- 2F5: A phage peptide library was screened with MAb 2F5, and from the peptides that bound the amino acids DKW were found to be most critical for binding – the mimetic peptide RDWSFDRWSLSEFWL elicited a cross-reactive Ab response to gp41 when used to immunize rabbits [Tumanova (2001)]
- 2F5: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding [York (2001)]
- 2F5: This paper primarily concerns 4E10 and Z13, MAbs that both bind proximally to the 2F5 binding site to a conserved epitope, and that neutralize some primary isolates from clades B, C, and E – the minimal 2F5 epitope is determined to be EQELLELDKWASLW, based on screening a gp160 fragment expression library, longer than previous studies – broadly neutralizing MAbs 2F5, IgG1b12, and 4E10 and Z13 fail to neutralize different subsets of viruses [Zwick (2001b)]
- 2F5: Neutralization synergy between anti-HIV NAbs b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two to four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 [Zwick (2001c)]
- 2F5: Review of studies in macaques that have shown immune control of pathogenic SHIV viremia, improved clinical outcome, and protection, and the implications of the observations for HIV vaccines [Mascola & Nabel(2001)]

- 2F5: SF162ΔV2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter and delivered by gene gun, SF162ΔV2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162ΔV2, but not intact SF162, was used as the immunogen – Control MAbs 2F5 and 2G12 could neutralize all of the following primary isolates: 91US056(R5), 92US714(R5), 92US660(R5), 92HT593(R5X4), and BZ167(R5X4), while after the first protein boost, the sera from two SF162ΔV2 immunized macaques could neutralize 91US056(R5), 92US714(R5), 92US660(R5) and ADA(R5), but not 92HT593(R5X4) or 92US657(R5) – the pattern of cross-recognition shifted after the second boost [Barnett (2001)]
- 2F5: Moore and colleagues review the data concerning the lack of a clear relationship between genetic subtype and serotype – 2F5 is considered in some detail, as it represents a rare vulnerability from the neutralizing antibody perspective, although while it is apparently linear, attempts to present the peptide to the immune system have failed to elicit neutralizing Abs [Moore (2001)]
- 2F5: Neutralizing synergy between MAbs 1b12, 2G12 and 2F5 was studied using surface plasmon resonance to determine the binding kinetics for these three mAbs with respect to monomeric and oligomeric env protein gp160 IIIB – the 2G12 epitope is highly accessible on both monomeric and oligomeric Envs, 1b12 is highly accessible on monomers but not oligomers, and 2F5 on neither form – binding of 2G12 exposes the 2F5 epitope on gp160 oligomers [Zeder-Lutz (2001)]
- 2F5: Matrix-assisted laser desorption ionization mass spectrometry (MALDI-MS) in combination with proteolytic protection was used to identify the functional epitope for MAb 2F5, NEQELLELDKWASLWN, in the disulfide bond associated gp120/gp41 protein SOS-gp140 (JRFL) – this minimal epitope is much larger than the ELDKWA core epitope previously defined by peptide ELISA, and this could help explain why ELDKWA-peptides are poor immunogens in terms of eliciting a 2F5-like antibody response [Parker (2001)]
- 2F5: A luciferase-reporter gene-expressing T-cell line was developed to facilitate neutralization and drug-sensitivity assays – luciferase and p24 antigen neutralization titer end points were found comparable using NAb from sera from HIV+ donors, and MAbs 2F5, 2G12 and IgG1b12 [Spenlehauer (2001)]
- 2F5: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 2F5: 4E10 binds proximal to 2F5 and neutralizes primary isolates of clades A, B, C, D, and E – viruses that were resistant to 2F5 were neutralized by 4E10 and vice versa [Stiegler (2001)]
- 2F5: A combination of MAbs IgG1b12, 2F5, and 2G12 was given postnatally to four neonate macaques that were then challenged with highly pathogenic SHIV89.6P – one of the four infants remained uninfected after oral challenge, two infants had no or a delayed CD4(+) T-cell decline [Hofmann-Lehmann (2001)]
- 2F5: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]
- 2F5: 2F5 or sCD4-IgG chimeric immunoadhesin were transferred into 3T3 cells, incorporated into a collagen structure called the neo-organ, and transplanted into SCIDhu mice that were then challenged with MN or LAI – the continuous production of the therapeutic molecules in this context resulted in dramatic reduction of viral load [Sanhadji (2000)]

- 2F5: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – 2F5 recognized o-gp140 [Srivastava (2002)]
- 2F5: UK Medical Research Council AIDS reagent: ARP3063
- 2F5: NIH AIDS Research and Reference Reagent Program: 1475

---

635 polyclonal gp160(662–667) gp41(662–667) ELDKWA; no Vaccine murine( )

Vaccine: *Vector/type: E. coli* MalE protein *HIV component:* gp41 peptide

References: [Coeffier (2000)]

- The antigenicity of ELDKWA inserted in MalE protein was estimated from 2F5 binding analysis using BIAcore(R) and its immunogenicity in mice was measured – specific but non-neutralizing MAbs were raised [Coeffier (2000)]

---

636 polyclonal gp160(662–667) gp41( ) ELDKWA L P Vaccine rabbit( )

Vaccine: *Vector/type:* peptide *HIV component:* gp41

Ab type: C-domain **References:** [Liao (2000)]

- Low levels of anti-ELDKWA antibodies are observed in HIV-1+ individuals, so a C-domain P2 peptide linked to a carrier was used to immunize mice and rabbits, and stimulated a high-level anti-ELDKWA response in mice and rabbits – vaccine was C-TSLIHSLIEESQNQQEKNEQELLELDKWA linked to carrier peptide K/G [(KGGG)_7-K] [Liao (2000)]

---

637 polyclonal gp160(662–667) gp41(669–674) Vaccine mouse, rabbit( )

Vaccine: *Vector/type:* peptide *HIV component:* Env *Stimulatory Agents:* BSA

Ab type: C-domain **References:** [Xiao (2000b)]

- Strong epitope-specific neutralizing antibody responses were induced using a Env peptide bound to BSA, C(ELDKWAG)_4-BSA, but not full gp160 [Xiao (2000b)]

---

638 polyclonal gp160(662–667) gp41(662–667 BH10) ELDKWA L Vaccine murine(IgG,IgA)

Vaccine: *Vector/type:* influenza virus *Strain:* BH10 *HIV component:* gp41 peptide

Ab type: C-domain **References:** [Muster (1994), Muster (1995)]

- Sustained ELDKWA specific IgA response in mucosa of immunized mice [Muster (1995)]

---

639 polyclonal gp160(662–667) gp120(669–674) ELDKWA Vaccine rabbit(Ig)

Vaccine: *Vector/type:* polyepitope, protein *HIV component:* gp160 *Stimulatory Agents:* BSA

Ab type: C-domain **References:** [Lu (2000b), Lu (2000a)]

- High titer response to ELDKWA and RILAVERYLKD was observed upon vaccination with multiple-epitope vaccine CG-GPGRAFY-G-ELDKWA-G-RILAVERYLKD conjugated to BSA, with a weak response to GPGRAFY – immunization with CG-(ELDKWA-GPGRAFY)_2-K was also tried, yielding a strong Ab response to both ELDKWA and GPGRAFY – gp160 vaccination yielded strong Ab response but not to any of the peptides studied here [Lu (2000b), Lu (2000a)]

---

640 TH-Ab1 gp160(662–667) gp41(669–674) ELNKWA L and P Vaccine rabbit(IgG1)

Vaccine: *Vector/type:* peptide *Strain:* B clade TH936705 *HIV component:* gp41 *Stimulatory Agents:* Freund's adjuvant

Ab type: C-domain **References:** [Xiao (2000a), Dong (2001)]

- TH-Ab1: ELNKWA is an escape variant not recognized by the broadly neutralizing MAb 2F5, which recognizes the core epitope ELDKWA – Abs were raised against the peptide escape variant CGELNKWAGELNKWA linked to keyhole limpit carrier protein – these polyclonal antibodies, like the monoclonal antibody TH-Ab1 also raised to ELNKWA, could recognize ELDKWA and escape mutant peptide epitopes ELEKWA and ELDEWA [Dong (2001)]

---

641 5B2 gp160(662–668) Env(669–674 IIIB) ELDKWA Vaccine mouse(IgG)

Vaccine: *Vector/type:* peptide in keyhole limpet hemocyanin *Strain:* IIIB *HIV component:* gp41

Ab type: C-domain **References:** [Tian (2001)]

- 5B2: There is an RT specific Ab [Szilvay (1992)] and a gp41 specific Ab [Tian (2001)] both called 5B2
- 5B2: Peptides GPGRAFY and ELDKWA were conjugated to keyhole limpet hemocyanin and used to raise mouse MAbs – MAb hybridomas were generated with defined specificity – 5B2 and 9G11 bind to the peptide and to rgp41 [Tian (2001)]

---

642 9G11 gp160(662–668) Env(669–674 IIIB) ELDKWA Vaccine mouse(IgG)

Vaccine: *Vector/type:* peptide in keyhole limpet hemocyanin *Strain:* IIIB *HIV component:* gp41

Ab type: C-domain **References:** [Tian (2001)]

- 9G11: Peptides GPGRAFY and ELDKWA were conjugated to keyhole limpet hemocyanin and used to raise mouse monoclonal Ab – MAb hybridomas were generated with defined specificity – 5B2 and 9G11 bind to the peptide and to rgp41 [Tian (2001)]

---

643 4E10 gp160(671–676) gp160(671–676 MN) NWFDIT P HIV-1 infection human(IgG3κ)

Donor: Herman Katinger, Inst. Appl. Microbiol. University of Agricultural Science, Vienna, Austria

References: [Buchacher (1992), Buchacher (1994), D'Souza (1994), Stiegler (2001), Zwick (2001b), Zwick (2001c), Xu (2001)]

- 4E10: MAbs generated by hybridoma, electrofusion of PBL from HIV-1+ volunteers with CB-F7 heteromyeloma cells – also binds to MHC class II proteins – anti-class II Abs are only found in HIV-1 positive people – this paper maps 4E10's binding site to AEGTDRV, gp160(823–829), but the later Zwick *et al.* study in 2001 revised the epitope location [Buchacher (1994)]
- 4E10: Included in a multi-lab study for antibody characterization, binding and neutralization assay comparison [D'Souza (1994)]
- 4E10: 4E10 binds proximal to 2F5 and neutralizes primary isolates of clades A, B, C, D, and E – viruses that were resistant to 2F5 were neutralized by 4E10 and vice versa [Stiegler (2001)]
- 4E10: MAbs 4E10 and Z13 both bind proximally to 2F5 to a conserved linear epitope that has some conformational aspects – both bind to MN virions, bind weakly to infected cells in a manner that is not disrupted by sCD4 and neutralize some primary isolates from clades B, C, and E – maps minimal 4E10 epitope to NWFDIT, contrary to an earlier report – different strains were refractive to neutralization by broadly neutralizing Abs IgG1b12, 2F5, Z13 and 4E10 [Zwick (2001b)]

- 4E10: Neutralization synergy between anti-HIV NAbs b12, 2G12, 2F5, and 4E10 was studied – a classic fixed-ratio method was used, as well as a method where one Ab was fixed at a low neutralization titer and the other was varied – using primary isolates, a two-four fold enhancement of neutralization was observed with MAb pairs, and a ten-fold enhancement with a quadruple Ab combination – no synergy was observed with any MAb pair in the neutralization of TCLA strain HXB2 [Zwick (2001c)]
- 4E10: Twenty HIV clade C isolates from five different countries were susceptible to neutralization by anti-clade B MAbs in a synergistic quadruple combination of mAbs IgG1b12, 2G12, 2F5, and 4E10 [Xu (2001)]

---

644 Z13 gp160(671–676) gp41(671–676 MN) NWFDIT P HIV-1 infection human(IgG1κ)

Ab type: C-term **References:** [Zwick (2001b)]

- Z13: MAbs 4E10 and Z13 both bind proximally to 2F5 to a relatively conserved linear epitope that has some conformational aspects – both bind to MN virions, bind weakly to infected cells in a manner that is not disrupted by sCD4 and can neutralize some primary isolates from clades B, C, and E – Z13 was selected using an antibody phage display library with the MN gp41 peptide LLELDKWASLWNWFDITNWSW from an HIV infected donor who had an exceptionally broad NAb response – different strains were refractive to neutralization by broadly neutralizing Abs IgG1b12, 2F5, Z13 and 4E10 – epitope location noted here is by analogy to MAb 4E10 [Zwick (2001b)]; ————————

---

645 B30 gp160(720–734) gp41(720–734 BH10) HLPIPRGPDRPEGIE Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

Donor: George Lewis

References: [Abacioglu (1994)]

- B30: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

646 polyclonal gp160(724–745) gp41(731–752) PRGPDRPEGIEEEGGERDRDRS Vaccine murine(IgA,IgG2a)

Vaccine: *Vector/type:* Cowpea mosaic virus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Durrani (1998)]

- Comparison of intranasal and oral immunization of HIV-1 peptide expressed in a plant viral vector – intranasal gave the better response [Durrani (1998)]

---

647 41S-2 gp160(725–745) gp160(732–750) RGPDRPEGIEEEGGERDRDRS yes Vaccine murine(IgG2bκ)

Vaccine: *Vector/type:* peptide *HIV component:* gp41 *Stimulatory Agents:* keyhole limpet hemocyanin

References: [Hifumi (2000)]

- 41S-2: BALBc mice were immunized with gp41 peptide and a MAb specific for the peptide was generated – isolated MAb light chains displayed proteolytic activity towards the peptide epitope which may be due to a catalytic triad on light chain (Asp73, Ser76, and His79) – no catalytic activity was observed for the whole antibody [Hifumi (2000)]

648	447-52D (447/52-DII, 447-52-D, 447d, 447-52-D, 447-D, 447, 447D)	gp160(312–315)	gp120(MN)	GPXR		L	HIV-1 infection	human(IgG3λ)

Ab type: V3 **Donor:** Dr. Susan Zolla-Pazner, NYU Med Center NY, NY, or Cellular Products Inc, Buffalo, NY, USA

References: [Gorny (1992), Buchbinder (1992), Karwowska (1992b), Gorny (1993), Keller (1993), Cavacini (1993a), Spear (1993), Conley (1994a), Laal (1994), VanCott (1994), Gorny (1994), Moore (1994a), Sattentau(1995), Fontenot (1995), Saarloos (1995), Zolla-Pazner (1995), Zolla-Pazner & Sharpe(1995), Moore (1995a), Moore & Ho(1995), Forthal (1995), Jagodzinski (1996), Trkola (1996a), Sattentau(1996), D'Souza (1997), Binley (1997a), Fouts (1997), Hioe (1997), Boots (1997), Parren (1997b), Hill (1997), Gorny (1997), Inouye (1998), Mondor (1998), Smith (1998), Parren (1998a), Zolla-Pazner (1999a), Zolla-Pazner (1999b), Connor (1998), Gorny (1998), Nyambi (1998), Hioe (1999), Beddows (1999), Gorny (2000), Grovit-Ferbas (2000), Hioe (2000), Ly & Stamatatos(2000), Nyambi (2000), Park (2000), York (2001), Verrier (2001), Srivastava (2002)]

- 447-52D: Requires GPXR at the tip of the V3 loop – neutralizes a broad array of B clade lab isolates [Gorny (1992)]
- 447-52D: 60-fold increase in neutralization potency when combined 1:1 with human MAb 588-D [Buchbinder (1992)]
- 447-52D: Reacts with MN, NY5, CDC4, SF2, RF, WM52, and HXB2 [Karwowska (1992b)]
- 447-52D: Neutralizes MN and IIIB: GPGR, and binds SF2: GPGR [Gorny (1993)]
- 447-52D: Peptide phage library showed that any of the residues ADGLMNQRS in the X position tolerated in peptides that react well with the antibody [Keller (1993)]
- 447-52D: Additive neutralization of MN and SF2 when combined with CD4 binding site MAb F105 – supra-additive neutralization of RF [Cavacini (1993a)]
- 447-52D: Complement mediated virolysis of IIIB, but not in the presence of sCD4 [Spear (1993)]
- 447-52D: Requires GPxR at the tip of the V3 loop, common in B clade – neutralized primary isolates [Conley (1994a)]
- 447-52D: Neutralization synergy in combination with CD4 binding domain MAbs [Laal (1994)]
- 447-52D: GPGQ in MAL resulted in enhanced dissociation – GPGQ in CM234 or K14T did not bind – binding affected by identity of amino acids flanking GPGR core [VanCott (1994)]
- 447-52D: Mild oxidation of carbohydrate moieties does not alter binding [Gorny (1994)]
- 447-52D: Competition studies with human sera from seroconverting individuals showed that anti-CD4 BS antibodies can arise very early in infection, comparable or prior to anti-V3 antibodies [Moore (1994a)]
- 447-52D: Called 447d – Formalin inactivation of virus at 0.1% formalin for 10 hours at 4 degrees was optimal for inactivation of virus while maintaining epitope integrity [Sattentau (1995)]
- 447-52D: Called 447 – The tip of the V3 loop was presented in a mucin backbone – higher valency correlates with stronger affinity constant [Fontenot (1995)]

- 447-52D: Ab-mediated activation of complement on HIV+ cells is higher than Ab independent activation – what has been termed "Ab independent" in fact results in part from IgM in normal human serum that is HIV-cross-reactive [Saarloos (1995)]
- 447-52D: Serotyping study using flow-cytometry – bound only to GPGR V3 loop tips [Zolla-Pazner (1995)]
- 447-52D: Neutralization of primary and prototype laboratory HIV-1 isolates using a resting cell assay enhances sensitivity [Zolla-Pazner & Sharpe(1995)]
- 447-52D: Binding affected by identity of amino acids flanking GPGR core – poor breadth of primary virus neutralization [Moore (1995a)]
- 447-52D: Review: the V3 loop motif GPGR is not common outside subtype B isolates, MAb 19b is more cross-reactive [Moore & Ho(1995)]
- 447-52D: Neutralizing (- complement), no ADCC activity, and no viral enhancing activity [Forthal (1995)]
- 447-52D: Called 447-52-D – The sulfated polysaccharide curdlan sulfate (CRDS) binds to the Envelope of T-tropic viruses and neutralizes virus – CRDS inhibits binding [Jagodzinski (1996)]
- 447-52D: Neutralizes JR-FL – strongly inhibits gp120 interaction with CCR-5 in a MIP-1β-CCR-5 competition study [Trkola (1996a)]
- 447-52D: Review: called 447-52-D – only four epitopes have been described which can stimulate a useful neutralizing response to a broad spectrum of primary isolates, represented by the binding sites of MAbs: 447-52-D, 2G12, Fab b12, and 2F5 [Sattentau(1996)]
- 447-52D: In a multilaboratory blinded study, failed to consistently neutralize any of nine B clade primary isolates – many of these isolates had the GPGR motif at the apex of the V3 loop [D'Souza (1997)]
- 447-52D: Study shows neutralization is not predicted by MAb binding to JRFL monomeric gp120, but is associated with oligomeric Env binding – 447-52D bound monomer, oligomer, and neutralized JRFL [Fouts (1997)]
- 447-52D: Tested using a resting cell neutralization assay [Hioe (1997)]
- 447-52D: Viral binding inhibition by 447-D was correlated with neutralization (all other neutralizing MAbs tested showed some correlation except 2F5) [Ugolini (1997)]
- 447-52D: Neutralizes TCLA strains but not primary isolates [Parren (1997b)]
- 447-52D: Called 447 – gp120 can inhibit MIP-1α from binding to CCR5, but this inhibitory effect is blocked by pre-incubation of gp120 with three anti-V3 MAbs: 447, 257, 1027 – MAb 670 which binds in the C5 region had no effect [Hill (1997)]
- 447-52D: Abs that recognize discontinuous epitopes can identify mimotopes from a phage peptide display library – 447-52D has an epitope involving the tip of the V3 loop, that was previously studied with this method [Keller (1993)] – in Keller *et al*., with no competition, LxGPxR was the most common six-mer, 38% of the peptides – after competition with a gp120 IIIB ligand (QRGPGR)i, RGPxR was the most common and one peptide had the sequence QRGPGR, showing type specific mimotyopes can be enriched by strain specific ligand competition protocols [Boots (1997)]
- 447-52D: Used as a control for comparison to five V3 RF selected antibodies – 447-52D was reactive with A, B, and C clade peptides, but not E [Gorny (1997)]
- 447-52D: Called 447-D – 447-D resistance took longer to acquire in virus with the M184V substituted RT, and had the form (AAC N to TAC Y) at position 5 of the V3 loop, rather than the GPGR to GPGR resistance found with wildtype RT [Inouye (1998)]
- 447-52D: Inhibits binding of Hx10 to both CD4 positive and negative HeLa cells [Mondor (1998)]
- 447-52D: Called 447-52-D – The tip of the MN V3 loop was inserted into cold causing human rhinovirus 14 (HRV14) – chimeras were immunoselected, and chimeric viruses were neutralized by anti-V3 loop antibodies, and 447-52D was among the Abs used – chimeric viruses elicited potent NAbs in guinea pigs against ALA-1 and MN [Smith (1998)]

- 447-52D: The MAb and Fab binding to the oligomeric form of gp120 and neutralization were highly correlated – authors suggest that neutralization is determined by the fraction of Ab sites occupied on a virion irrespective of the epitope [Parren (1998a)]
- 447-52D: Ab from gp120 vaccinated individuals prior to infection, who subsequently became HIV infected, could not achieve 90% neutralization of the primary virus by which the individuals were ultimately infected – these viruses were not particularly refractive to neutralization, as determined by their susceptibility to neutralization by MAbs 2G12, IgG1b12, 2F5 and 447-52D [Connor (1998)]
- 447-52D: Kinetic parameters were measured, and the association rates were similar, but dissociation rate constants were quite variable for V3 MAbs, 1324E was comparable to 447-52D [Gorny (1998)]
- 447-52D: Using a whole virion-ELISA method, 18 human MAbs were tested for their ability to bind to a panel of 9 viruses from clades A, B, D, F, G, and H – 447-52D was the most potent and cross-reactive of 18 human MAbs tested and was the only MAb which bound to virions from isolates CA20 (subtype F), CA13 (subtype H), and VI526 (subtype G) [Nyambi (1998)]
- 447-52D: Review of clade specificity and anti-V3 HIV-1-Abs [Zolla-Pazner (1999a)]
- 447-52D: MAb peptide-reactivity pattern clustered with the immunological related MAbs: 1334, 419, 504, 447, 453 and 537 – the core amino acids GP tended to be critical for reactivity in this group – 447 reacted with peptides containing GPGR, but also with many lacking this sequence (GPGQ, for example), and it failed to react with 2/14 peptides containing GPGR, illustrating the importance of context [Zolla-Pazner (1999b)]
- 447-52D: The presence of leukocyte function-associated molecule 1 (LFA-1) promotes virus infectivity and hinders neutralization, and anti-LFA-1 MAbs can enhance the neutralizing effect of anti-HIV V3 MAb 447-52D and anti-HIV CD4BS MAb IgG1b12 – non-neutralizing anti-HIV CD4BS MAb 654-D did not become neutralizing in the presence of anti-LFA-1 MAbs [Hioe (1999)]
- 447-52D: rgp120 derived from a R5X4 subtype B virus, HIV-1 W61D, was used to vaccinate healthy volunteers and the resulting sera were compared with sera from HIV-1 positive subjects and neutralizing MAbs – TCLA strains showed enhanced 447-52D neutralization sensitivity relative to PBMC-adapted lines (32X increase between HIV-1(M2424/PBMC(p0)) and HIV-1(M2424/H9(p9)) and a >128X increase between HIV-1(W61D/PBMC) and HIV-1(W61D/SupT1) isolates) [Beddows (1999)]
- 447-52D: Binding of a panel of 21 MAbs to soluble oligomeric gp140 was compared to gp41 and gp120 monomers – no MAb was oligomer specific, though anti-V3 and CD4BS MAbs reacted better with the oligomer and V2 and C5 tended to favor the monomer – V3 MAbs 447-52D, 838-D, and 1334 bound with a 7–10 fold preference for the oligomer [Gorny (2000)]
- 447-52D: To determine the antigenicity of virus killed by thermal and chemical inactivation, retention of conformation-dependent neutralization epitopes was examined, and exposure of CD4BS epitopes was found to be enhanced (MAbs IgG1b12, 205-46-9, and 205-43-1) – binding to 2G12 and 447-52D epitopes was essentially unaltered – the 17b CD4i epitope was also exposed [Grovit-Ferbas (2000)]
- 447-52D: Ab responses, because of their capacity to alter antigen uptake and processing, can influence helper T cell responses – CD4BS MAbs or serum Ig from HIV+ individuals inhibited proliferative responses of gp120 specific T cells – V3 MAbs 447-52-D and 268–10-D did not effect proliferation [Hioe (2000)]
- 447-52D: Called 447D – SF162 is a neutralization-resistant HIV-1 isolate – N-linked glycosylation modifications in the V2 loop of the SF162 gp120 revealed that these sites prevent neutralization by CD4BS MAbs (IgG1b12 and IgGCD4), and protect against neutralization by V3 MAbs (447D and 391–95D) – V2-region glycosylation site mutations did not alter neutralization resistance to V2 MAbs (G3.4 and G3.136) or CD4i MAbs (17b and 48d) – V2 glycosylation site modification allows infection of macrophages, probably due to glycosylated forms requiring fewer CCR5 molecules for viral entry [Ly & Stamatatos(2000)]

- 447-52D: A panel of 47 human MAbs was tested against 26 HIV-1 group M primary isolates from clades A through H – 19 V3 MABs were tested, and of 494 combinations, 44% displayed some viral binding – V3 MAbs tended to have the most cross-reactive binding to clade A, B, C, and D isolates, less to E, F, G, and H – 447-52D showed the highest cross-reactivity, bound to 24/26 viruses tested, but achieved 90% neutralization only against MN, 50% against CA5, and no neutralization was observed for 3 other isolates tested [Nyambi (2000)]
- 447-52D: Six mutations in MN change the virus from a high-infectivity neutralization resistant phenotype to low-infectivity neutralization sensitive – V3, CD4BS, and CD4i MAbs are 20–100 fold more efficient at neutralizing the sensitive form – the mutation L544P reduced binding of all MAbs against gp120 by causing conformational changes [Park (2000)]
- 447-52D: Abs against the V3 loop (50.1, 58.2, 59.1, 257-D, 268-D, 447-52D), CD4BS (IgG1b12, 559–64D, F105), CD4i (17b), and to gp41 (2F5, F240) each showed similar binding efficiency to Env derived from related pairs of primary and TCLA lines (primary: 168P and 320SI, and TCLA: 168C and 320SI-C3.3), but the TCLA lines were much more susceptible to neutralization suggesting that the change in TCLA lines that make them more susceptible to NAbs alters some step after binding – the dissociation constant, Kd of 447-52D for the cell associated primary and TCLA Envs was equal, 3nM [York (2001)]
- 447-52D: A panel of 12 MAbs was used to identify those that could neutralize the dual-tropic primary isolate HIV-1 89.6 – six gave significant neutralization at 2 to 10 μg/ml: 2F5, 50–69, IgG1b12, 447-52D, 2G12, and 670-D – six did not have neutralizing activity: 654-D, 4.8D, 450-D, 246-D, 98–6, and 1281 – no synergy, only additive effects were seen for pairwise combinations of MAbs, and antagonism was noted between gp41 MAbs 50–69 and 98–6, as well as 98–6 and 2F5 [Verrier (2001)]
- 447-52D: Oligomeric gp140 (o-gp140) derived from R5 primary isolate US4 was characterized for use as a vaccine reagent – antigen capture ELISA was used to compare the antigenicity of gp120 and o-gp140 using a panel of well characterized MAbs – 447-D recognized the gp120 monomer much more readily than the o-gp140, suggesting the V3 loop is less exposed on o-gp140 as it is on the intact virions [Srivastava (2002)]

---

649 C8 gp160(727–732) gp41(727–732 BH10) PDRPEG no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

References: [Pincus & McClure(1993), Pincus (1993), Abacioglu (1994), McLain (2001)]

- C8: Immunotoxin of C8 coupled to ricin-A does not mediate cells killing, and is not affected by sCD4 [Pincus & McClure(1993)]
- C8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – C8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus (1993)]
- C8: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]
- C8: The substitution R725G (P[R→G]GPDRPEGIEEEGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

---

650 B31 gp160(727–734) gp41(727–734 BH10) PDRPEGIE Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

References: [Abacioglu (1994)]
- B31: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

651 B33 gp160(727–734) gp41(727–734 BH10) PDRPEGIE no Vaccine murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* NL43 *HIV component:* gp160

References: [Abacioglu (1994), Bristow (1994)]
- B33: There are two MAbs in the literature named B33, see also gp120, positions 123–142 – MAb generated in a study of the humoral immune response to Baculovirus-expressed mis-folded rgp160 IIIB:NL43, MicroGenSys [Bristow (1994)]
- B33: Epitope boundaries mapped by peptide scanning IgG1 [Abacioglu (1994)]

---

652 1576 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Vella (1993)]
- 1576: Not neutralizing [Vella (1993)]

---

653 1578 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Evans (1989), Vella (1993)]
- 1578: No neutralizing activity – epitope may be formed by regions from both poliovirus and HIV [Evans (1989)]
- 1578: Core epitope: IEEE – in this study, neutralized IIIB, but not RF or MN [Vella (1993)]

---

654 1579 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Vella (1993)]
- 1579: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)]

---

655 1583 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Evans (1989), Vella (1993), Sattentau (1995)]
- 1583: Neutralizing activity, less broad than 1577 [Evans (1989)]
- 1583: Core epitope: ERDRD – Could neutralize HIV IIIB but not HIV RF [Vella (1993)]
- 1583: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)]

---

656 1899 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Vella (1993)]
- 1899: Could neutralize HIV IIIB and HIV RF [Vella (1993)]

---

657 1907 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Vella (1993)]

- 1907: Could not neutralize HIV IIIB, RF or MN [Vella (1993)]

---

658 1908 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Evans (1989), Vella (1993), Sattentau (1995)]

- 1908: Neutralized IIIB, but not RF or MN [Vella (1993)]
- 1908: Cytoplasmic domain, epitope not exposed at the surface of HIV-1 infected cells [Sattentau (1995)]

---

659 1909 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

References: [Vella (1993)]

- 1909: Neutralized HIV IIIB but not HIV RF [Vella (1993)]

---

660 41–1 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine(IgMκ)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *HIV component:* gp41 peptide

References: [Mani (1994), Dalgleish (1988)]

- 41–1: This antibody gp41(735–752 IIIB) [Dalgleish (1988)] seems to have been named the same as a different MAb to gp41(584–609) [Mani (1994)]
- 41–1: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]

---

661 41–2 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine(IgMκ)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *HIV component:* gp41 peptide

References: [Dalgleish (1988)]

- 41–2: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]

---

662 41–3 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no Vaccine murine(IgMκ)

Vaccine: *Vector/type:* peptide *Strain:* IIIB *HIV component:* gp41 peptide

References: [Dalgleish (1988)]

- 41–3: Neutralizes HIV-1 but not HIV-2 strains [Dalgleish (1988)]

---

663 ED6 gp160(728–745) gp41(735–752 IIIB) DRPEGIEEEGGERDRDRS no murine(IgM)

References: [Evans (1989)]

664	LA9 (121–134)	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no		murine(IgM)

References: [Evans (1989)]

665	1575	gp160(728–745)	gp41(735–752 IIIB)	DRPEGIEEEGGERDRDRS	no	Vaccine	murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

Ab type: C-term **Donor:** C. Vella, NIBSC, Potters Bar UK
References: [Evans (1989), Vella (1993), Buratti (1997), Cleveland (2000a)]

- 1575: Neutralizing activity, less broad than 1577 [Evans (1989)]
- 1575: Core epitope: IEEE – neutralized IIIB, but not RF or MN [Vella (1993)]
- 1575: Study shows that MAb 1575 can recognize the IEEE sequence in both gp41, and in the HPG30 region of the p17 protein – motif is conserved in both regions in different HIV-1 clades [Buratti (1997)]
- 1575: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)]

666	88–158/02	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR	Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp41

References: [Niedrig (1992a)]

- 88–158/02: Mild inhibition of *in vitro* activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)]

667	88–158/022	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR	Vaccine	murine(IgG2b)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp41

References: [Niedrig (1992a)]

- 88–158/022: Mild inhibition of *in vitro* activity at high MAb concentrations – profound enhancing activity at low concentrations – significant reactivity to virion – domain non-immunogenic in humans [Niedrig (1992a)]

668	88–158/079	gp160(732–747)	gp41(732–752 IIIB)	GIEEEGGERDRDRSIR	Vaccine	murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* IIIB *HIV component:* gp41

References: [Niedrig (1992a)]

- 88–158/079: Mild inhibition of HIV *in vitro* at high MAb concentrations – profound enhancing activity at low concentrations – weak binding to virion – domain non-immunogenic in humans [Niedrig (1992a)]

669	polyclonal	gp160(dis 733–736)	gp41(dis 735–752 IIIB)	IEEE	L	Vaccine	murine(IgG)

Vaccine: *Vector/type:* Cowpea mosaic virus *HIV component:* gp41 peptide

Ab type: C-term **References:** [Cleveland (2000b), McLain (2001)]

- When PRGPDRPEGIEEEGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD [Cleveland (2000b)]

- The substitution 725 RG (P[R–>G]GPDRPEGIEEEGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

---

670 polyclonal | gp160(dis 733–736) | gp41(dis 735–752 NL43) | IEEE | L | Vaccine | murine(IgG)

Vaccine: *Vector/type:* Cowpea mosaic virus *HIV component:* gp41 peptide

Ab type: C-term **References:** [McLain (2001)]

- The substitution 725 RG (P[R–>G]GPDRPEGIEEEGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

---

671 B8 | gp160(733–741) | gp41(733–741 BH10) | IEEEGGERD | no | Vaccine | murine(IgG1)

Vaccine: *Vector/type:* recombinant protein *Strain:* LAI *HIV component:* gp160

References: [Pincus (1993), Abacioglu (1994)]

- B8: Ab response in IIIB lab workers was compared to gp160 LAI vaccine recipients – B8 was used as a control – the dominant response among vaccinees was to this mid-gp41 region, but not among the infected lab workers – Abs binding this region do not neutralize, bind to infected cells, nor serve as immunotoxins [Pincus (1993)]
- B8: Epitope boundaries mapped by peptide scanning [Abacioglu (1994)]

---

672 1577 | gp160(739–743) | gp41(735–752 IIIB) | ERDRD | no | Vaccine | murine( )

Vaccine: *Vector/type:* poliovirus *Strain:* IIIB *HIV component:* gp41 peptide

Ab type: C-term **Donor:** C. Vella or Morag Ferguson (NIBSC, Potters Bar UK)
References: [Evans (1989), D'Souza (1991), Vella (1993), Cleveland (2000a)]

- 1577: Raised against IIIB peptide chimera – neutralized African and American HIV-1 lab strains [Evans (1989)]
- 1577: Non-neutralizing in this multi-lab study [D'Souza (1991)]
- 1577: Core epitope: ERDRD – could neutralize HIV IIIB and HIV RF [Vella (1993)]
- 1577: Ab binding to IEEE suppresses neutralizing Ab binding to adjacent epitope ERDRD [Cleveland (2000a)]
- 1577: UK Medical Research Council AIDS reagent: ARP317
- 1577: NIH AIDS Research and Reference Reagent Program: 1172

---

673 polyclonal | gp160(dis 739–743) | gp41(dis 735–752 IIIB) | ERDRD | L | Vaccine | murine(IgG)

Vaccine: *Vector/type:* Cowpea mosaic virus *HIV component:* gp41 peptide

Ab type: C-term **References:** [Cleveland (2000b), McLain (2001)]

- ERDRD-specific IgG recognizes an externalized loop of the gp41 C-terminal tail with high affinity – neutralized HIV-1 B clade strains IIIB, NL-4.3, RF, MN and D clade virus CBL-4, but HXB-2D (clade B) was not recognized – when PRGPDRPEGIEEEGGERDRDRS was used as antigen an immunodominant, non-neutralizing response to IEEE was observed, but immunization GERDRDR shifts the response to ERDRD – NAb does not inhibit attachment of free virus, but does inhibit an event that precedes fusion-entry [Cleveland (2000b)]
- The substitution 725 RG (P[R–>G]GPDRPEGIEEEGGERDRDRS) alters the antigenic exposure of this region on the virion resulting in the loss of the downstream neutralizing epitope ERDRD, increased exposure of the epitope GPDRPEG in the virion, while the epitope IEEE remains unchanged [McLain (2001)]

---

674 DZ | gp160(822–855) | gp41(827–860 BRU) | VAEGTDRVIEVVQGACRAIRH-IPRRIRQGLERIL | L | Vaccine | human(IgG1λ)

Vaccine: *Vector/type:* vaccinia *Strain:* IIIB *HIV component:* gp60

References: [Boyer (1991)]

- DZ: Weakly neutralizing IIIB – binds to peptides 827–843 and 846–860 of BRU – reacted specifically with IIIB and RF [Boyer (1991)]

---

675 IVI-4G6 | gp160( ) | gp41( ) | Vaccine | murine(IgG2b)

Donor: K. Miyakoshi (Feji-Rebio Co, Tokyo, Japan)

References: [Yin (2001)]

- IVI-4G6: A bi-specific Ab (BFA) was made by combining Fab fragments of gp41-specific MAb IVI-4G6 and CD3-specific Mab UCHT1 – the BFA suppressed HIV-1 propagation culture and eliminated latently infected cells [Yin (2001)]

---

676 polyclonal | gp160( ) | gp120( ) | no | Vaccine | mouse( )

Vaccine: *Vector/type:* recombinant protein, virus-like particle *Strain:* LAI *HIV component:* V3, CD4BS, p55

Ab type: CD4BS **References:** [Truong (1996)]

- Antibodies raised against recombinant anti-p55 virus-like particles with the p24 region 196–226 deleted, bearing inserts of either the V3 or the CD4BS regions of gp120 were studied – no neutralizing responses, weak Env and strong Gag responses were elicited – the major homology region (MHR) and proximal regions were found to be required for capsid assembly [Truong (1996)]

---

677 polyclonal | gp160( ) | gp120( ) | no | Vaccine | mouse( )

Vaccine: *Vector/type:* recombinant protein, virus-like particle *Strain:* LAI *HIV component:* V3, CD4BS, p55

Ab type: V3 **References:** [Truong (1996)]

- Antibodies raised against recombinant anti-p55 virus-like particles with the p24 region 196–226 deleted, bearing inserts of either the V3 or the CD4BS regions of gp120 were studied – no neutralizing responses, weak Env, and strong Gag responses were elicited – the major homology region (MHR) and proximal regions were found to be required for capsid assembly [Truong (1996)]

678	polyclonal	gp160( )	gp140(SF162)	KSITIGPGRAFYATGD	yes	Vaccine	rabbit, Rhesus macaque(IgG)

Vaccine: *Vector/type:* DNA, CMV promotor elements *Strain:* SF162, SF162ΔV2 *HIV component:* gp140 *Stimulatory Agents:* MF-59C

Ab type: V3 **References:** [Barnett (2001)]

- SF162ΔV2 is a virus that has a 30 amino acid deletion in the V2 loop that does not abrogate its infectivity but renders it highly susceptible to neutralization – when incorporated into a codon-optimized DNA vaccine with a CMV promoter, delivered by gene gun, SF162ΔV2 gave higher neutralizing Ab titers against SF162 than did SF162 itself, and Abs that cross-neutralized non-homologous primary isolates were obtained only when SF162ΔV2, but not intact SF162, was used as the immunogen – NAbs titers specific for SF162 increased with multiple immunizations, while titers for non-homologous isolates decreased, but anti-V3 peptide binding Abs were not likely the source of this distinction because anti-V3 titers were much lower than those against the entire envelope, and the second booster immunization did not increase the titer of anti-V3 loop Abs [Barnett (2001)]